New Orleans Medical
and
Surgical Journal

Vol. 92 JULY, 1939 No. 1

DECEASED MEMBERS OF THE LOUISIANA STATE MEDICAL SOCIETY*

"O COME AND MOURN WITH ME AWHILE"

S. J. COUVILLON, M. D.

MOREAUVILLE, LA.

I accepted with no small degree of appreciation, the honor in having been chosen the "Memorial Speaker" at so auspicious an occasion—The Memorial Exercises, conducted annually by the State Medical Society at a public session of its annual convention, designed for the general public as well as the bereaved families and members of the medical profession in commemoration of the untimely passing of every member of the Healing Art in the last twelve months within the confines of Louisiana.

To the thirty-four members of the Louisiana medical profession, in whose memory we are assembled this evening, to eulogize and pay proper homage, must our attention be turned. Our medical heroes, dead or alive, are never remembered by the people as are the heroes and soldiers, dead and alive, in the way of public praises and ovations following and during conflicts of warfare, nor the engulfed admiration of movie sirens as they make short stop-overs in some of our heavily populated centers. Such honors never befall the silent, tireless and consistent laboring doctor while fighting the battle with life and death, day in and day out, whether it be in the immaculate rooms of a hospital or in the humble home of some poor worker, until at a time following so many deaths we gather in this modest way, with bowed heads, with sad hearts, to memorialize our dead confrères. I do not believe that there is a quotation or verse more fitting, more appropriate, and so true as that of our literary friend, Thomas Gray, in his "Elegy Written in a Country Churchyard," when he said:

> "The boast of heraldry, the pomp of power,
> And all that beauty, all that wealth e'er gave,
> Awaits alike the inevitable hour—
> The path of glory leads but to the grave."

Death is sad, very sad, and while it may seem that the cloak of modesty is being ignored, yet it seems impossible to refrain from saying that death is particularly sad when the Grim Reaper snatches away the life of an element of our people whose mission on earth, and whose devotion to duty, are designed not only in the prolongation of the lives of the people, but when possible, the prevention of death; that element of human intelligence with a trained conception of medical and surgical knowledge who night and day wage a terrific fight, not only against a visible enemy, but against an enemy unseen, unheard; against nature and against death. Said a great American physician at the recent session of the American College of Physicians in New Orleans, "scientific knowledge cannot be dispensed in boxes and crates as a market commodity, but knowledge can only emanate from skilled hands, schooled and keen minds, honest hearts."

Yes, death is sad, and doubly sad because it is just the commencement of another

*Memorial address read before the sixtieth annual meeting of the Louisiana State Medical Society, April 24, 1939.

struggle—an endless struggle so beautifully versed by the great poet:

> "Think thee my soul
> That death is but a Groom,
> Which brings a taper
> To the outward room."

The thirty-four good doctors, most of whom were members of the State Medical Society, whose deaths we are assembled here this evening to chronicle and share like sorrows, were physicians and surgeons actively engaged in their respective callings in the last year, and I point with satisfaction that most of them were known to me. I point with distinction and regret to the passing of one of my two remaining college professors, Dr. Abraham Louis Metz, two clinical instructors, five class-mates, one room-mate, several close medical and political friends, two Avoyelles Parish confrères, Dr. Barbin and Dr. Poret, who died within a few hours of each other, buried the same day in nearby towns—one funeral throng going from one to the other—a coincidence, but such a sad epoch; several college fraternity brothers, a few railway physician and surgeon associates. Many of the deceased group were friends as a result of acquaintances made at various medical conventions and assemblies. In reviewing this sad roll of dead physicians, with a glance we note the ages they had attained, ages yet filled with such pronounced activities and usefulness to their country, and yet realize, as George Hebert so plainly, so sadly versed, saying that their:

> "Flesh is but the glass, that holds the dust,
> That measures our time, which also shall
> Be crumbled into dust."

And yet, in the Elysian lands above, these thirty-four medical warriors have joined other souls, hand in hand, shoulder to shoulder, heart to heart—all for the everlasting common cause—reviewing their earthly labors so often performed without any thought of compensation, so often prompted by the love of their sacred calling, and the satisfaction they were rewarded in the realization of a work well done. This is the picture and status of the medical profession with its many ideals, unparalleled in all other professions, trades or vocations. It is universally recognized that we are actually the only class of professional people who so often work without remuneration. Think of the many charity hospitals of the country, the so many other eleemosynary institutions, medical leagues, medical health units, the various public health departments of the land which are conducted only by the medical and surgical men of a country with such tasks and responsibilities attached. All of this carries with it little, if any, compensation, and all of this is regardless of the many sacrifices we are daily called upon to do in private practice, when the so many indigent sick and helpless, who are all as human as any of us, demand our services. At this time I believe it is befitting to quote the phraseology of Voltaire, the French philosopher and writer, who said: "But nothing is more estimable than the physician who, having studied nature from his youth, knows the properties of the human body, the diseases which assail it, the remedies which will benefit it, exercises his art with caution and pays equal attention to the rich and the poor." Think also of the little thought which is being given to the number of years the doctor must devote to his preparations, regardless of the financial aspect, before he is capable of handling the lives of humanity—years of young manhood and womanhood, studiously bent over textbooks, absorbed in the various laboratories, with the years slipping away while he reaches and searches for all the knowledge and skill which it is humanly possible to acquire, years when other young men and women are enjoying their youth, or are branching out into some other field of adventure, years when the medical student is immersed in the deep mysteries of the human body. Again we realize the public spiritedness, the civic energy which has always been in the minds and hearts of our doctors. Will you agree with me that lay minds understand that the medical man has always been very frank and honest with the public at large? The bridging of the gap between the modernized

version of herb medicines, the chemical laboratory with its sufanilamides and pyridines, the x-ray with its so many technics of diagnosis and therapy, are all being well perfected and the world is daily being told about the various accomplishments, propagated, as it were, to the lay people in intelligent articles, private lectures and radio talks, mediums which are aiding materially in casting aside the prejudices so common in ancient times, when our people in all faith would crowd around the village street corner at the banjo music and comedy acts of a medicine show and bought what the showman offered, just as his savage ancestors had given devout heed to the chants and antics of their wizards. The old medicine show is almost as rare now as the "dodo"; the quack nostrum is on the wane, but the atmosphere has been and is still full of medical heroes and heroines. Yes, full of martyrs, of immortals, of everlasting sacrifice, usefulness, such as has been the careers of Professors Elliott, Metz, Lewis; of Doctors Seemann, Dowling, Cazenavette, Hume, Roy, Wilson, Robin, Meraux, Gremillion, Dupuy, Fenno, Cappel, Ducote, Signorelli, and the many excellent practitioners and community leaders of our country, men who always towered high in the estimation of those who value rectitude and have the proper appreciation for the lofty attributes that make life honorable and beautiful. And while all sympathy centers on the thirty-four bereaved families of Louisiana this evening, yet the honor and the glory, sayeth John Dryden, poet and prose writer, is theirs:

> "Tis best to die,
> Our honor at the height."

May I ask that you stand with me with bowed heads as I read the list of the deceased in the last year:

Walter H. Brent, Hammond.
Aristides T. Barbin, Marksville.
Isadore Brickman, Alexandria.
Lionel L. Cazenavette, New Orleans.
Frederick L. Fenno, New Orleans.
Gaston L. Gaudet, Lutcher.
John A. Hendrick, Shreveport.
John Raymond Hume, New Orleans.
Louis A. Meraux, New Orleans.
Caroline Mims, New Orleans.
Edward A. Poret, Hessmer.
Robert H. Potts, New Orleans.
William H. Robin, New Orleans.
J. Alphonse Sampite, Cloutierville.
William H. Seemann, New Orleans.
John Signorelli, New Orleans.
Augustus E. Simonton, Jonesboro.
Davidson K. Texada, Alexandria.
Herbert B. Wren, Shreveport.
Albert S. Yenni, New Orleans.
Robert D. Voorhies, Lafayette.
H. P. Doles, Mooringsport.
R. E. Ellzey, Cotton Valley.
Frank J. Hartley, New Orleans.
Clinton E. Hunter, Jennings.
William L. Litton, Cheneyville.
Orrin F. Matthews, Urania.
Abraham Louis Metz, New Orleans.
Sam H. McMahon, DeRidder.
James P. Powell, Mangham.
John E. Rooks, Shreveport.
John B. Sewell, St. Francisville.
John Tolson, Church Point.
Sidney LeG. Williams, Shreveport.

"Eternal rest grant unto them, O Lord, and let perpetual light shine upon them."

—o—

SOME LOUISIANA UROLOGISTS

HENRY W. E. WALTHER, M. D.
NEW ORLEANS

The history of urology as a specialty is a development of the twentieth century, but urology as a field of study, is one to which many of the great physicians and surgeons for several centuries have devoted much time and attention. Louisiana has contributed her just share to the advancement of this branch of surgery during the past seventy years. In gathering data concerning the story of the growth of knowledge on urology in Louisiana, I have examined the files of four medical journals founded in New Orleans, namely, L'union médicale de la Louisiane, New Orleans Medical News and Hospital Gazette, New Orleans Medical Times, and New Orleans Medical and Surgical Journal. This last, the second oldest medical journal in the United States, is the

only one of the four that survives today. In pursuing the pages of these chronicles one discovers an amazing mass of material in evidence of the interest in urology as a field of study for the years covered, namely, 1844 to 1914. The story of such an epoch naturally concerns itself with the men who made it possible. One hundred and sixty-eight articles on urologic subjects by physicians of Louisiana have appeared in the above named medical journals, within the dates named, 54 of which were written by eleven men, the majority of said contributions having appeared before the turn of the century. Enumerated by subjects they include: 33 papers each on the bladder and on the urethra, 17 on urine and urinary disorders, 16 on kidney, 14 on the testes, 13 on urinary fistulas, 12 each on hydrocele and penis, nine on the prostate, five on the scrotum and four on the hypospadias.

As an example of the interest shown in genito-urinary diseases in Louisiana, in the early days, I quote from the Southern Medical and Surgical Journal for 1846 as follows: "Prize Essays — The Louisiana Medico-Chirurgical Society offers a gold medal of the value of one hundred dollars, for the best Essay on "Strictures of the Urethra, with Their Treatment." This prize is offered to the competition of the profession in all countries; but the essays must be written in the English or French language. The communications must be accompanied with a letter of corresponding mottoes, to the President of the Louisiana Medico-Chirurgical Society, New Orleans, La., and should be received by the first of February, 1847."

The considered infrequency of calculous diseases in the South, in the early literature, is worthy of particular mention. In 1896, G. B. Johnston, after a study on the subject of stone in the bladder in the white and negro races, wrote, "Some states are peculiarly exempt from calculous disease, notably, Mississippi, Arkansas, Alabama, Louisiana and Florida." The same year, Rudolph Matas, in his "Surgical Peculiarities of the American Negro", said, "Urinary calculus is a comparatively rare disease in both races, in the whole territory that borders on the Gulf Coast . . . According to our experience in the Charity Hospital it would appear that urinary calculus is less frequent but more fatal in the negro than in the white." With the introduction of cystoscopy and x-ray these opinions had to be modified.

It is gratifying to find that the list of authors includes such giants in the history of medicine in Louisiana as Warren Stone, Alexander J. Wedderburn, Anthony Peniston, Isaac L. Crawcour, Tobias G. Richardson, Edmond Souchon, Samuel Logan, Andrew W. Smythe, Rudolph Matas, Charles Chassaignac, Frederick W. Parham, Carroll W. Allen and C. Jeff Miller.

Isaac L. Crawcour was the first in Louisiana to devote special attention to so-called urinary diseases. In the medical register and directory of the United States edited by Dr. Butler (2d ed.—1878) appears the entry:

> Crawcour, Isaac L., 251 Camp St., M. D.; Medical School of N. O.; Prof. Med. N. O. Med. Sch.; Corresponding Member of Society of Arts, England;—specialty, Diseases of the Urinary Organs.

Crawcour was born in Devonshire, England, in 1825. He studied medicine in London, became a resident in Guy's Hospital and graduated from the University of London, we are told, in 1851, coming to New Orleans in December of that year. He received an honorary degree from New Orleans School of Medicine where he became professor of medical chemistry in 1858. He was one of the founders of the New Orleans Academy of Sciences, of which he was an active member for many years. His obituary in 1866 says, he "was a man of the highest intelligence and great accomplishments in medicine . . . The city is indebted to him for the first introduction of some instruments and methods of procedure now in general use." Crawcour's articles, all of which preceded 1869, include reports on diseases of the urinary and genito-urinary organs, constitutional effects of substances introduced into the urethra and on the urine and urinary deposits. He maintained a laboratory in his home and in his later

years made a particular study of nephritis. He was undoubtedly a real pioneer in his field.

Another type of specialist who preceded the urologists was the physician who devoted a large part of his time to the treatment of venereal diseases. Among these in New Orleans were Edgar S. Poincy and Adrian Hava. Of Poincy little has been learned except that he graduated from the Medical College of Louisiana in 1881 and practiced in New Orleans in the eighties. Hava was born in 1865, coming to New Orleans from Havana. He graduated from the Medical College of Louisiana in 1881, earning a degree as doctor of pharmacy as well as that of doctor of medicine from this school. Hava maintained an extensive practice in the city, although living for many years in Waveland, Mississippi. He died in 1933.

We are indebted to Edmond Souchon for two articles on urethral spasm, one on double hydrocele and one on the philosophic anatomy of the kidney—during the period studied. Souchon was a native Louisianian, having been born in Opelousas in 1842. He received his medical training in Paris, and shortly after graduation served as interpreter for J. Marion Sims, when Sims visited Paris to demonstrate his operation for vesico-vaginal fistula. Souchon returned to New Orleans and was engaged in medical work in this city for nearly sixty years. He was Vice-President of the American Medical Association and of the American Surgical Association. He was a founder of the Southern Surgical Association and was an Honorary Fellow of the American College of Surgeons. He served Tulane University as professor of anatomy from 1884 to 1907; was emeritus professor of anatomy from 1907 until his death. In his honor, the University, in 1908, created the Souchon Museum of Anatomy, as a perpetual memorial to his forty years of devotion to the department he had so ably directed. He died in 1924.

Charles Chassaignac was the first physician in Louisiana to limit his practice to urology. Chassaignac was born in New Orleans in 1852. He graduated from the Medical Department of the University of Louisiana, now Tulane University of Louisiana, in 1883, and practiced general medicine in his native city for several years, after which he went to France for study and research. Returning to New Orleans, he associated himself with the New Orleans Polyclinic, later the Post-Graduate School of Tulane University School of Medicine, serving as dean from 1898 to 1925. He occupied the first chair of genito-urinary diseases in this school, serving from 1890 to 1925. His practice was limited to genito-urinary diseases from 1895 to his death. He was president of the Orleans Parish Medical Society and of the Louisiana State Medical Society and edited the New Orleans Medical and Surgical Journal from 1896 to 1922, first in collaboration with Isadore Dyer and later alone. He started the Genito-Urinary Out-Patient Clinic at Charity Hispital in 1913. He was one of the founders of the New Orleans Sanitarium and Training School for Nurses. Although Rudolph Matas was the first in America to use spinal anesthesia (at the New Orleans Charity Hospital in 1899), Chassaignac was the first to sponsor its use in genito-urinary surgery in Louisiana. He died in 1936.

Sidney P. Delaup was for some years an associate of Chassaignac in genito-urinary practice. Delaup was born in 1863. He graduated from Tulane University School of Medicine in 1890, after which he continued his studies in Philadelphia, New York, London, Paris, and Berlin. Returning to New Orleans, he served in the Genito-Urinary Out-Patient Clinic of Charity Hospital with Chassaignac. From 1893 to 1905, he was clinical assistant in genito-urinary diseases in the New Orleans Polyclinic and from 1905 until his death in 1923, he served as professor of surgery of the genito-urinary organs and rectum in this school. In 1910, Delaup reported 1,239 operations under spinal anesthesia, most of them urologic procedures. This was the largest tabulation at the time and probably the first dealing with spinal anesthesia in urology in the United States.

Paul J. Gelpi, also on the staff of the New Orleans Polyclinic, was born in New Orleans in 1874. He graduated from the Tulane University School of Medicine in 1896, after which he studied in France, Germany and Austria. He was made clinical assistant in genito-urinary and rectal diseases of the Polyclinic in 1899, later being made lecturer, then assistant professor and professor, serving until 1925. Gelpi died in 1932.

Abraham Nelken, who practiced urology in New Orleans for some years, was born in 1876. He graduated from Tulane University School of Medicine in 1899. Nelken published four articles on genito-urinary subjects in local journals between 1904 and 1913. He was chief of the Urological Division of Touro Infirmary for fifteen years, until his death in 1926.

A truly colorful figure in the history of urology in Louisiana was Carroll W. Allen, who was born in 1874. A general surgeon by vocation, he made urology and local anesthesia his hobbies. His masterful textbook of 674 pages dealing with "Local and Regional Anesthesia", first published in 1914, remains to this day a standard reference. In this book appeared, for the first time, his description of suprapubic prostatectomy performed under local infiltration anesthesia with novocain. He was a pioneer in this field. As the years passed it was his desire to relinquish surgical urology and devote himself solely to general surgery but until the last, patients from every corner of the country and from abroad came to him to have the prostate removed under local anesthesia. His gentleness, his patience and his sympathetic understanding endeared him to his many assistants as well as to his patients. For many years he was assistant professor of clinical surgery in Tulane University Medical School; not a few of the men practicing urology throughout the South today owe much to the lectures, clinical demonstrations and operative seminars enjoyed at Charity Hospital under Allen. He served the Southern Baptist Hospital as chief of the Surgical Division from 1926 until the time of his death in 1934.

Particularly noteworthy in this group of New Orleans urologists was Joseph Hume, who was born in Summerville, South Carolina, in 1876. He was educated in the schools of Charleston, and the University of Pennsylvania, and was an honor student in the Medical College of South Carolina. Following his graduation he went to Johns Hopkins University where he studied his chosen specialty under the direction of Hugh Hampton Young. Hume came to New Orleans in 1904 and in 1905 became lecturer and clinical instructor in genito-urinary and venereal diseases in Tulane University School of Medicine; in 1927 he was made professor of urology and from 1931 to 1935, he served as director of the Division of Urology. He became professor emeritus of urology on his resignation in 1935 because of ill health. Hume died in 1936.

Thus, Louisiana has more than kept pace with the rest of the United States, both in the study of the genito-urinary tract and in the development of urology as a specialty. New Orleans, in this, as in other branches of medicine is one of the country's centers, for study and medical research, and the city's urologists are proud of their heritage—the pioneer work of those who have gone before.

The writer wishes to acknowledge his indebtedness to Miss Mary L. Marshall for her valued collaboration in collating the data embodied in this tribute.

—o—

SCLERODERMA*

A SYMPOSIUM

THE DERMATOLOGIC ASPECTS

JAMES KIRBY HOWLES, M. D.†

NEW ORLEANS

When this paper was first conceived it was my intention to summarize the dermatologic and laboratory findings in eight cases of generalized scleroderma which had come under my observation during the last ten years. Later, when this symposium was planned and I was asked to discuss the dermatologic features of this bizarre

*Read before the Orleans Parish Medical Society, January 23, 1939.

†From the Division of Dermatology and Syphilology of the School of Medicine of Louisiana State University.

and interesting disease, it seemed wiser to omit the case reports, to avoid trespassing on the fields assigned to the other essayists of the evening. An apology is in order for the necessarily elementary and "textbook" character of my presentation.

The first authentic case of scleroderma seems to have been reported by Curzio of Naples in 1752.[1] There was little interest in the condition, and only occasional cases were reported until 1845, when Thirial recorded two cases under the name "Sclérème des adultes." Thereafter the disease was reported with increasing frequency. It is, however, still an unusual and misunderstood condition. The literature is now replete with case reports and reports of research work, yet scleroderma remains an enigma to the dermatologist.

The multiplicity of names by which scleroderma is described tend to confuse the picture. Perhaps the most common dermatologic term is "hidebound skin," which vividly pictures the rigidity and loss of elasticity of the involved skin in both the localized and diffuse types. Other synonyms used by English-speaking dermatologists include scleroderma adultorum, morphea (localized scleroderma), scleriasis, dermatosclerosis, chorianitis, and sclerostenosis. The foreign nomenclature is even more inclusive and confusing.

CLASSIFICATION

Sellei's[2] classification, although it is accepted by many European dermatologists as ideal, is too inclusive to be really practical:

1. Scleroderma verum.
2. Acrosclerosis.
   a. Sclerodactylia.
3. Morphea (en plaque).
   a. Morphea guttata.
4. Scleroderma adultorum (Buschke).
   a. Sclerema neonatorum.
5. Acrodermatitis atrophicans.

A simpler clinical classification will be used as the basis of this discussion:

1. Circumscribed scleroderma (morphea).
2. Diffuse scleroderma.
3. Sclerodactylia.
4. White spot disease (Westberg's[3] disease, morphea guttata).

ETIOLOGY

The exact etiology and pathogenesis remain unknown. The most reasonable theories of origin include:

1. *A toxic origin.* The champions of this theory believe that some preexisting disease or toxic focus precipitates the pathologic change, although the exact nature of the focus remains undetermined. Such inflammatory conditions as erysipelas, certain liver infections and certain rheumatic states have been suggested. Cornbleet and Struck[2] consider that scleroderma is caused by a toxin which injures the collagen synctium, and that the injured tissues take up calcium secondarily.

Sannicandro[4] advances a bacillary origin. He supports his diagnosis of sarcoidal sclerodermic tuberculosis by three arguments, the response to gold therapy, the positive cutaneous tuberculin reaction, and the histologic findings in certain forms of scleroderma. Milbradt[5] considers infection of the liver the etiologic factor on the basis of telangectasia hemorrhagica hereditaria (Osler's syndrome) which he found associated with scleroderma in a single case. Whitehouse[6] advances coexistent infection as a cause, particularly syphilis, and Ayers[6] believes that drug intoxication, particularly by arsenic, may be responsible.

2. *Glandular and metabolic dysfunction.* This hypothesis is the subject of a great deal of controversy. Thus Sequeira[2] attributes the disease to dysfunction of the adrenal glands, but Foerster[2] considers pathologic changes in the thyroid gland as the underlying etiologic factor. The most reasonable theory is that the parathyroid glands are responsible, for the most promising advance in the therapy of scleroderma has been achieved by parathyroidectomy. Hypercalcemia is not a constant finding, but has been noted in a sufficient number of cases to be correlated with a chronic disturbance of the parathyroid metabolism. Brooks,[7] in reporting one case of calcinosis and another of scleroderma, thoroughly reviewed the literature, and abnormal calcium

and phosphorus values seem numerous enough in the reported cases to warrant continued study along this line.

3. *Pathologic changes in the nervous system.* There are many advocates of neurologic disorders as the etiologic basis of scleroderma, and it is impossible at this time to disprove many of these theories. Hoffman[2] believes that the disease is caused by instability of the vegetative nervous system. Murchison[6] has reported degenerative lesions of the spinal cord in association with it. Sequeira[2] believes that the pathology of morphea or localized scleroderma lies in the central ganglia rather than the peripheral nerves. On the other hand, Kinqery[6] and Aronstam[6] have reported interesting cases of scleroderma associated with or subsequent to trauma to the peripheral nerves.

After literally wading through these and other hypotheses, one is inclined to believe with Castle[6] that scleroderma is due to disordered function of certain glands of internal secretion in combination with some disorder of the nervous system. Practically all of the theories which have been advanced are applicable in certain instances, but a theory which will cover all cases has not yet been formulated.

HISTOPATHOLOGY

Most dermatologists, according to Ormsby,[2] consider scleroderma to be a trophoneurosis or angiotrophoneurosis. He quotes Osler, as follows: "The analogy of myxedema, to which scleroderma is the cutaneous antithesis, suggests that it may be caused by some alteration in an internal secretion, or some disturbance of that nice balance between the various internal secretions . . . which some believe play such an important role in nutrition . . . The frequency with which acute forms follow an infection is paralleled by the thyroid insufficiency and atrophy caused by myxedema after a fever, such as measles and scarlet fever."

Ormsby continues: "The histologic changes of all clinical types of scleroderma are practically identical. In the stage of infiltration there occurs marked hypertrophy of the collagenous tissues through the corium and hypoderm, which is cell-rich and associated with vascular dilatation, causing effacement of the normally wavy line made by the alternating rete pegs and papillae. At this point the stage of atrophy sets in, the connective tissue gradually becoming more and more sclerotic, thinning the corium and subcutis, the fat disappearing, the cellular infiltration remaining only in small perivascular areas, the blood vessels becoming obliterated from pressure and from inflammation of their walls, most of them, like the hair follicles, sweat and sebaceous glands, later becoming absorbed. The epidermis is thinned by pressure atrophy, and the elastic tissue is mechanically altered, but seldom to the point of marked destruction. There may be an increase of pigment present in either stage."

A brief summary of histopathologic alterations will include the following: (1) Scleroderma is a chronic inflammatory process of the skin and subcutaneous tissue; (2) there are two stages, the early edematous and late atrophic; (3) the inflammatory infiltrative changes disappear promptly; (4) the histologic structure is essentially the same in all clinical forms; (5) the epithelium, except for thinning due to atrophy, is only slightly involved; (6) pressure atrophy destroys the hair follicles and glands; (7) the blood vessels become less numerous and their lumina are narrowed; (8) there is a perivascular lymphocytic infiltration; (9) hypertrophy of the collagenous tissue occurs; (10) papillae are obliterated by widening the connective tissue fibers; (11) fatty tissue is replaced by sclerotic tissue.

CLINICAL MANIFESTATIONS

The clinical manifestations of scleroderma are frequently perplexing, particularly when they coexist with other diseases in which atrophy and sclerosis may be part of the clinical picture. Such confusion may occur in Raynaud's disease, Addison's disease, and syphilis.

Circumscribed scleroderma (*morphea*) is usually limited to one or several patches on the skin, most often on the chest or abdomen. Most commonly they are round or oval, and slow-growing. The lesions may

be preceded for weeks or even for months by an itchy patch, red or violaceous in color. The initial patch gradually fades and is replaced by a sclerotic lesion which is glossy and indurated, like polished bone or ivory, and is surrounded by a well-defined violaceous or telangiectatic border, the color being due to dilated capillaries. The skin of the lesion is inelastic, its natural furrows disappear, and hair and sweat glands are destroyed. The consistency, as has been well said, suggests a bladder filled with lard. The follicular orifices may be unusually prominent, and eventually the lesion may resemble pigskin. Occasionally the plaques are slightly elevated and resemble keloids.

The localized form of scleroderma may progress to the diffuse variety, but more often the plaques remain stationary for months or years and then undergo regressive changes, either spontaneously or as the result of treatment. They may reassume the appearance of normal skin, or may remain as brownish patches without much change in the texture of the involved skin (morphea nigra). Atrophic changes may occur in other cases, or a hemiatrophy, either alone or in association with generalized scleroderma.

Localized scleroderma may also occur in the form of stripes or bands. Sometimes the two forms appear together. The striped variety is particularly common in children, and usually appears on the limbs. The bands may be single or multiple. They vary in width from a quarter to half an inch or more, and may be of considerable length. They may follow the course of a rib, or the longitudinal axis of an extremity, or they may appear in the middle of the forehead.

Unna[3] describes a "card-like" scleroderma, in which the lesions have a bluish-white color and resemble a small portion of a visiting card let into the skin. The lesions are multiple, somewhat depressed, and much smaller than those of typical morphea.

Diffuse scleroderma is characterized by the appearance of multiple hard, smooth, ivory-colored areas, which are immobile and which are usually surrounded by a violaceous margin. The appearance varies according to the site of involvement and the stage and extent of the disease.

Early in the disease the affected areas are usually erythematous and edematous. The edema pits only on sustained deep pressure, and is soon replaced by atrophy. The skin becomes smooth, yellowish, and firm, and is so retracted that the underlying structures are bound down. The changes occur insidiously, usually involving the face and hands, and in more advanced stages these parts become literally "hidebound," and the face is absolutely expressionless. Warty, pigmented, blackish patches may appear about the elbows and neck, and in the axillae and groins. The disease usually begins in the upper part of the body, especially on the neck and shoulders, but may also begin on the face, trunk, or extremities. It usually spreads in a more or less symmetrical fashion.

During the progress of the disease the pressure of the hard overlying skin brings about atrophy of the muscles and bones, often with resultant deformity. The limbs become so shrunken that flexion is impossible. The lips become thin and tight, the nose shrivelled, as in late lupus, the eyelids retracted, and the eyeballs shrunken, with a blank, staring expression. Sclerotic changes in the ear drum may cause deafness. The mucous membrane may be involved, and the trophic patches appear on the inside of the cheeks, and on the palate and tongue. The teeth may become loose and fall out. The mucous membrane of the vagina also may be affected.

The vitality of the affected skin is so lowered that even slight injuries, especially about prominent parts like the elbows and knees and the malleolar region, are likely to be followed by breaks in continuity, intractable ulceration, and even local gangrene. Late in the disease hyperpigmentation or depigmentation or a diffuse bronzing of the skin may be present, and the affected areas may become hairless.

In rare instances diffuse scleroderma may be complicated by the presence of subcutaneous calcareous deposits which are apparently a terminal phase of the fibrosis. They originate in the midst of dense con-

nective tissue, gradually reach the surface, and there, after ulceration has ensued, are finally expelled. Chemically these concretions consist of calcium carbonate and calcium phosphate. They occur most frequently about the joints.

Diffuse scleroderma, although subject to intermissions, is slowly progressive. It may not reach its full development for months or years. It may regress without sequelae, may remain stationary, or may progress until death occurs, usually from bronchopneumonia or some other intercurrent infection.

Sclerodactylia is a variety of diffuse scleroderma which begins on the hands and tends to spread to the arms, neck, face, and chest. It attacks several fingers of one hand, or attacks both hands more or less symmetrically. Progressive sclerotic changes occur, and ulceration and cicatrization are occasionally found, especially on the bony prominences of the knuckles or joints of the fingers. The nails become dystrophic, discolored, or opaque, and are sometimes narrowed and distorted, or completely obliterated. The fingers become hard, stiff, and shiny, and the skin is gray. They may be permanently distended or semi-flexed because of ankylosis of the joints. The terminal phalanges may undergo spontaneous necrosis and absorption and the fingers may be transformed into shrivelled stumps. All grades of disfigurement may be present, from a claw-like deformity resembling the "main griffe" of leprosy to a mutilated extremity with atrophic stumps.

Morphea guttata (*white spot disease, Westberg's disease*) is characterized by multiple, rounded, atrophic macules, which are sometimes surrounded by a purplish-blue zone or by a halo of various colors. The lesions occur chiefly in clusters or lines on the upper part of the chest and neck, but the arms and other regions may also be involved. The spots are chalk-white and shiny, and range in diameter from 1 to 10 mm. They sometimes coalesce and form bizarre plaques, and the surface may be covered with telangiectases, scales, or follicular dots. Sometimes the lesions are slightly elevated and resemble small keloids. Old lesions may become hyperpigmented. Eventually all lesions become atrophic and slightly depressed, and are covered with a thin, parchment-like epidermis. MacKee and Wise[3] believe that all reported cases of white spot disease can be divided into two groups, a true scleroderma group and a lichen group (lichen planus sclerosis).

It is evident from what has been said that all varieties of scleroderma are characterized by extreme induration and rigidity of the skin, with ultimate atrophic changes. Indeed, atrophy is the outstanding feature of the disease. It seems most reasonable to assume in the light of our present knowledge that it is chiefly due to interference with the nerve supply and to glandular dysfunction.

SYMPTOMATOLOGY

Scleroderma develops insidiously, with mild and variable symptoms. It may appear without prodromal symptoms of any sort. In the localized variety itching may be the prodromal symptom. The diffuse variety may be preceded by some general disturbance such as fever, chilliness, or pains in the joints or limbs. This variety may also develop acutely, with associated erythema, urticaria, or bullous formation, but this is not usual.

The lesions are usually insensitive themselves. The first definite symptom is a feeling of tightness or stiffness of the affected area, which eventually prevents freedom of motion and ultimately any motion at all. There may be stiffness of the neck, difficulty in opening the eyes or mouth, and rigidity of the limbs. Later it may be impossible to masticate the food or open the mouth, and even respiratory motion may be restricted.

When the extremities are involved rheumatoid arthritis is fairly common. In sclerodactylia vasomotor paroxysms occur and increase in frequency until a permanent asphyxial phase ensues. There is considerable pain associated with the early crises, and later ulceration is responsible for pain, particularly when there is an attempt to move the rigid joints. Pain is not so marked when the lesions begin on the lower extremities.

DIFFERENTIAL DIAGNOSIS

A well marked circumscribed scleroderma can scarcely be mistaken for any other affection, for the flat, ivory-white, circumscribed, violet-rimmed unilateral patches are characteristic. The stage of the disease as well as the type of the eruption are, however, responsible for more confusing and more varied manifestations. Completely to differentiate the four varieties of scleroderma which have been discussed would require almost a textbook. It must suffice here merely to mention the important conditions which must be ruled out to establish diagnosis. The differentiation includes:

From diffuse scleroderma: Dermatomyositis, erythromelalgia, myxedema, acrodermatitis chronica atrophicans, sclerema, edema neonatorum, icthyosis, and xeroderma pigmentosum.

From sclerodactylia: Leprosy, syringomyelia, and Raynaud's disease.

From morphea (circumscribed scleroderma): Leprosy, cicatrix, and epithelioma.

From white spot disease: Lichen planus atrophicans and vitiligo.

THERAPY

There is no panacea for scleroderma, and treatment is, on the whole, entirely unsatisfactory. Time seems to be the chief factor in the healing of all varieties of the disease. No local measures have proved of any value, nor have general measures, though they are naturally desirable, as an improved general condition will always improve a local state.

Ormsby[2] gives an excellent summary of the various forms of therapy which have been recommended. Roques recommends small doses of thyroid extract given over long periods of time, and others have reported apparent improvement with it. It should be begun promptly. Little demonstrated a patient suffering with scleroderma associated with Graves' disease and later myxedema, who had been greatly benefited by the internal administration of thyroid extract and the implantation of human thyroid gland into the bone marrow. Suprarenal extract has been of value in a small number of cases, according to Ormsby, but pituitary extract and ovarian and testicular extracts are of doubtful value.

Ferments of various kinds have been recommended. Sellei recommends pancreatic ferments. Kolle used coeliacin, an extract of the mesenteric gland, and Schwerdt believes that it has merit. Liver extract has been used tentatively but is of very doubtful value. Vitamin D therapy has become popular in recent years. Reports are still few, but Cornfleet and Struck recommend it in daily doses of 200,000 to 300,000 international units.

Tonics are indicated, as the general health of the patients is usually impaired; those containing iron or iodine are most popular. The internal administration of thiosinamin, salol, ammonium chloride, pilocarpine, the salicylates, and acetyl-chlorine has also been recommended.

Topical therapy plays an important part in lubricating the skin and helping to overcome the dryness and hardness present in advanced stages, but otherwise is of no value. The most useful agents are cod liver oil ointment, lanolin, neat's foot oil (scented) and plasters.

The results of physical therapy are neither consistent nor impressive. Epstein[2] has used thorium X emanations in morphea, incorporated in an ointment base containing eucerine, 1000 to 2000 electrostatic units per gram. The inconvenience and expense of this method are not justified by the reported results.

A great deal has been written concerning the eradication of foci of infection as a curative measure in scleroderma. Obviously when a multiplicity of foci are present, the elimination of some of them will enable the patient better to tolerate those which remain. To me this is the explanation of many so-called cures, not only in scleroderma but in other diseases.

A change of climate, especially removal to a warmer climate, is of temporary benefit, as is dietary regulation. All symptomatic treatment may be helpful, but the results are always short-lived and the recalcitrance of the disease promptly becomes evident again.

The contributions of Leriche and his coworkers[8] in the field of surgical therapy bid fair to revolutionize the treatment of scleroderma. The remarkable results which have been achieved in the Strasbourg clinic indicate that experience and skill underlie them. Because Dr. DeBakey has worked with Leriche it is unnecessary for me to discuss this phase of therapy, except to say that I have been most impressed with his and Dr. Ochsner's results. In such work as theirs seems to lie the chief hope for patients suffering with scleroderma.

SUMMARY

1. Scleroderma may be classified clinically as circumscribed (morphea), diffuse, sclerodactylia, and white spot disease.

2. The etiology remains unknown, though dysfunction of the parathyroid glands is the most reasonable hypothesis, perhaps with an associated neurologic basis.

3. The histopathology, clinical manifestations, and symptomatology of the various forms are discussed.

4. The typical manifestations of the disease are readily diagnosed. Less typical forms must be differentiated from many other dermatologic states and from diseases with dermatologic complications.

5. Therapy is still unsatisfactory, but surgery, as developed in the Leriche clinic, offers the most hopeful outlook.

REFERENCES

1. Boardman, W. P.: Scleroderma with special reference to its etiology and treatment, Arch. Dermat. & Syphilol., 19:901, 1929.

2. Ormsby, O. S.: A Practical Treatise on Diseases of the Skin for the Use of Students and Practitioners, Ed. 5. Lea and Febiger, Philadelphia, 1937.

3. Pusey, W. A.: The Principles and Practice of Dermatology Designed for Students and Practitioners, Ed. 4. D. Appleton and Company, New York and London, 1926.

4. Sannicandro, G.: Sarkoide und sclerodermatige Tuberkulose mit symmetrischen Streifen an den Beinen, verbunden mit progressiver Sklerodermie, Arch. f. Dermat. u. Syph., 175:623, 1937.

5. Milbradt, W.: Lupus erythematodes acutus und thrombopenische Purpura; zugleich Bemerkungen über das Blutbild beim Lupus erythematodes, Dermat. Wchnschr., 105:997, 1937.

6. Sutton, R. L.: Diseases of the Skin, Ed. 8. C. V. Mosby Company, St. Louis, 1931.

7. Brooks, W. D. W.: Calcinosis, Quart. J. Med., 3:293, 1934.

8. Leriche, R., Jung, A., and DeBakey, M.: The surgical treatment of scleroderma, Surgery, 1:6, 1937.

ETIOLOGY AND ABNORMAL PHYSIOLOGY

GEORGE E. BURCH, M. D.†
NEW ORLEANS

It is only within recent years that intensive studies have been directed toward the subject of peripheral vascular diseases. This has been partly due to the development of more accurate methods for studying peripheral vascular phenomena as well as the increase in the number of individuals suffering from these diseases since the span of human life has been extended. Of the peripheral vascular diseases, scleroderma has been interesting to physiologists and clinicians not only because of its peculiar clinical characteristics but also because of its rarity. Its incidence has been variously stated. It occurs approximately once out of every 8,000 general clinic patients, or once out of every 1,000 dermatologic patients.[1] The clinical syndromes, pathologic features, course, and other phases of scleroderma are fairly well known. On the other hand, its etiology, pathologic physiology and therapy are little understood. The discussion to follow is an attempt to evaluate the etiologic and physiologic aspects of the disease and to point out the value of objective methods in its study.

ETIOLOGY

As in all diseases of unknown etiology, innumerable factors have been advanced to explain the cause of scleroderma. A number of the more important factors concerned with the etiology of this disease will be discussed.

ENDOCRINE

Practically all of the glands of internal secretion have been considered a primary or contributing cause of scleroderma.

Thyroid: The thyroid was one of the earliest and most frequently accused of the endocrine glands.[2] The effects of the thyroid are not consistent, however. Hypofunction as well as hyperfunction have

†From the Department of Medicine, School of Medicine, Tulane University of Louisiana, New Orleans, La.

been said to explain the relationship of thyroid dysfunction with the onset of scleroderma.[3] Osler[4] was not convinced that the eight patients he treated with thyroid extract were definitely benefited. Roques[5] collected from the literature 67 cases in which thyroid therapy was used, and found that four were cured, 32 benefited, and 31 unaffected. In 31 cases of diffuse scleroderma, it was found on physical examination that the thyroid was small or imperceptible in 14 instances, enlarged in eight and normal in nine. O'Leary and Nomland[2] failed to find a basal metabolic rate below normal in any of their cases of diffuse scleroderma. In seven cases there was evidence of hyperthyroidism and in one typical exophthalmic goiter. Castle[6], in a study of the literature on the endocrine causation of scleroderma, was impressed by the frequency with which the thyroid appeared as a factor etiologically influencing scleroderma, although he thinks the other glands of internal secretion are significant factors as well. Again, as stated by Stokes in discussing Longcope's paper,[7] scleroderma is associated with either hypo- or hyperthyroidism. He doubts that thyroid extract is specific, but thinks it may be beneficial through the production of vasodilatation and flushing. Oliver and Lerman[8] also attribute the benefit obtained from thyroid therapy to its secondary effect of increasing the cutaneous circulation. Dercum[9] suggested that the improvement produced by thyroid is probably dependent upon its action on subcutaneous fat, since it materially hastens the atrophy and disappearance of this tissue in the atrophic stage of the disease. Brown, O'Leary and Adson[10] feel that the sclerosis found in the thyroid gland at necropsy is a systemic effect of the scleroderma rather than the cause of it. Aub et al,[11] found the thyroid gland to influence calcium metabolism and still not necessarily produce changes in serum calcium, and also found hypothyroidism to be associated with a decrease in the excretion of calcium and phosphorus. These findings would tend to support the thyroid rather than the parathyroid as the cause of the calcium and phosphorus findings reported in scleroderma. This is discussed more fully below.

The evidence in the literature is far from convincing that the thyroid gland is primarily associated with the cause of scleroderma. What little benefit is derived from the use of thyroid extract apparently originates in its secondary effects on the circulation and metabolic processes. Of course, it is indicated to remedy a thyroid dysfunction that may be present in a patient suffering from scleroderma.

Parathyroid Glands and Calcium and Phosphorus Metabolism: The reason for including calcium and phosphorus metabolism with the discussion of parathyroid dysfunction is self-evident. Like the thyroid, the parathyroids have been said to be responsible, at least in part, for scleroderma.[8, 12-16] Selye,[17] while studying the effects of parathyroid extract on rats, found that young suckling rats developed within a few days a specific skin disease, which, on more careful study, was found to simulate human scleroderma. Shelling, Asher and Jackson[18] produced skin changes similar to those described by Selye in suckling rats following injections of parathormone. Leriche, Jung and Sureyya by means of injections of parathyroid extract were able to produce in young pigs skin changes which histologically resembled very closely the findings in human scleroderma. They noted that the calcium content of the skin was doubled or trebled after two or three injections. Hueper,[20] on the other hand, was able to produce calcification in the viscera but not in the skin of dogs following the injection of parathyroid extract. Leriche and Jung[15] are convinced that hyperparathyroidism is the cause of scleroderma and, therefore, as a therapeutic procedure remove most of the parathyroid tissue in patients suffering with this disease. They state that in scleroderma the parathyroid glands are the primary, the bones the intermediate, and the skin the final site of the disturbance in the chain of disturbed function. Leriche and his associates consider the disturbed

calcium and phosphorus metabolism reported in scleroderma to be due to hyperfunction of these glands. Bernheim and Garlock[14] removed most of the parathyroid tissue in six patients with Raynaud's disease and scleroderma, and found hypertrophy of the glands in every instance. The glands removed from one patient with extensive scleroderma showed acute parathyroiditis with pronounced infiltration with polymorphonuclear leukocytes. These authors think it is often necessary to obtain many serum calcium and phosphorus determinations before evidence of parathyroid dysfunction can be detected. They also do not consider hypercalcemia and hypophosphatemia necessary for surgical attack on the parathyroids. Furthermore, they consider the disturbed calcium metabolism the cause for the underlying vasospasm in scleroderma. Garlock[16] attributes the spasm of Raynaud's disease, which often precedes sclerodermatous change, to disturbed calcium metabolism. Hypertrophy, hyperplasia and adenomata have been found in the parathyroid glands removed at operation.[12, 14, 21] These changes, however, are not consistently encountered. It is interesting to note that Ssamarin, quoted by Cornbleet and Struck,[12] reported several cases in which supposed parathyroid tissue removed at operation was shown by microscopic examination not to be parathyroid and yet the patients experienced results equal to those in patients from whom actual parathyroid tissue was removed.

It has been shown[12, 13, 17, 19, 22, 23] that there is deposited in the sclerodermatous skin an increased amount of calcium, while the serum calcium of the same patients has been found to be normal or elevated.[12, 21, 24, 25] The serum phosphorus is either normal or elevated rather than decreased, as would be expected in true hyperparathyroidism.[12] Again, the few studies on urinary output of calcium have shown it to be markedly decreased,[12] while the commonest finding in hyperparathyroidism has been a negative calcium balance. The two reported studies of calcium balance in scleroderma showed in one instance a positive balance, while in the other a positive balance was found only when calcinosis was present. These findings are in agreement with those of Cornbleet and Struck.[12] There has been no evidence of generalized decalcification of bone, as is expected in hyperparathyroidism.[26] Leriche and Jung[15] report decalcification of bone but only describe changes in those of the hands and arms of the involved parts. This may be due to disuse and the local vascular changes and not to any generalized disturbance, as occurs in hyperparathyroidism.

Mamon[27] presents evidence supporting hypoparathyroidism in scleroderma. He attributes the tetany often associated with scleroderma, as well as other findings, to a low blood calcium and hypoparathyroidism. He was able to find in the literature reports of patients who developed scleroderma and signs of tetany following thyroidectomy. The author does believe, however, that there are cases in which evidence of hyperparathyroidism may be present. Such cases, he feels, deserve parathyroidectomy.

The association of calcinosis with scleroderma is well known.[12, 22, 26, 28] It is not known whether this calcification is primarily of parathyroid dysfunction, as discussed above, or of secondary origin unrelated to the parathyroid. Brooks[22] described a patient with calcinosis who showed evidence of calcium and phosphorus retention with normal absorption of these elements and abnormally high levels of serum calcium, plasma inorganic phosphorus and plasma phosphatase. He also described a patient with scleroderma in whom all these substances were normal. Brooks concludes that "in calcinosis a local tissue change, of as yet undetermined kind, results in localized fibrous tissue damage, and that when calcification occurs in these areas there may be an accampanying abnormality of calcium and potassium metabolism. The type of lesion present in scleroderma does not necessarily produce changes which result in abnormality in the metabolism of calcium and phosphorus." Brooks' paper is quite an extensive review of calcinosis.

Cornbleet and Struck[12] offer the hypothesis that scleroderma is initially due to a toxin which injures the collagen syncytium and that these injured tissues secondarily take up calcium. This deposition of calcium may account for the positive balance frequently seen in scleroderma. With massive doses of Vitamin D, these observers were able to increase the excretion of calcium and phosphorus in the urine, a negative balance produced apparently at the expense of the calcium deposited in the collagen and muscle. Acid drugs[29] and ketogenic diets[28] have also been employed to mobilize the deposited calcium, as well as to prevent a further deposition.

Durham[26] states that there are several mechanisms through which calcification might conceivably occur in association with scleroderma: (1) As a result of parathyroid dysfunction; (2) as metastatic calcification; (3) by simple chemical precipitation, and (4) by means of a physicochemical disturbance in which collodial proteins play a role. He points out the fact that "in experimental calcification produced by injecting parathyroid hormone, the deposits occurred most frequently in the excretory organs, the lungs, stomach and kidneys. The tissues of these organs are more alkaline because of their continuous excretion of acids. As the secretion from sudoriparous glands is normally neutral or slightly alkaline, and since the interglandular tissues around the sweat glands are slightly acid after the alkaline secretion, the occurrence of deposits of calcium in the skin or subcutaneous tissue as a result of parathyroid disturbances does not parallel experimental observations. Histologically, too, there is a difference. In the experimental type, the deposits of calcium were found in the interglandular tissue around the excreting glands, whereas in the reported cases with cutaneous and subcutaneous involvement, the deposits were not in the interglandular tissue, but were scattered diffusely throughout the connective tissue." Furthermore, the parathyroid glands are usually histologically normal. Durham again points out that rarefaction of skeletal bones is usually a constant occurrence in so-called metastatic calcification. In scleroderma with calcinosis there was no rarefaction except osteoporosis resulting from disuse. There was no oversaturation of the blood with calcium as is thought to occur in the metastatic variety of calcinosis. Trauma is usually necessary for this type of calcification. He concludes that it is, therefore, unlikely that the mechanism of calcification with scleroderma is similar to metastatic calcification. Durham finds the evidence insufficient to explain the calcinosis in scleroderma on the basis of local alterations or deranged inorganic metabolism, although, as in other types of pathologic calcification, the collodial proteins are thought to play a prominent role. He feels that local inflammations are usually not a causative factor. Durham, as well as Pautrier,[24] has suggested the possibility that calcium plays an important role in physicochemical phenomena in the interstitial colloids affecting the albuminous and mineral substances. The exact nature of this remains obscure in scleroderma.

Pituitary Gland: This gland has often been blamed for scleroderma,[5, 6, 8, 30, 31] but like the other endocrine glands there have been no consistent abnormal findings, clinical or histologic. Oliver and Lerman[8] state that the response of scleroderma to injections of posterior pituitary preparations is probably the result of peripheral circulatory effects rather than that of substitution. They treated 20 patients with scleroderma of different types with daily injections of posterior pituitary. In three cases of morphea some, or all, of the lesions disappeared completely, leaving only slight pigmentation, while only varying degrees of improvement were obtained in others. In two cases of the band-like type improvement was marked. Five patients with sclerodactylia associated with scleroderma of the face, arms, hands and neck, were definitely improved, while in three other similar cases some improvement was noted. The remainder of the 20 patients were helped by the injection. Schwartz, in discussing Fox's paper,[31] said he obtained good results with

the use of pituitary extract in six patients with scleroderma and an increased glucose tolerance.

The Other Endocrine Glands: The adrenals, [2, 3, 5, 6, 32, 33] gonads,[5, 8, 34] islands of Langerhans,[35] and thymus[22] have been blamed at various times for scleroderma. The pigmentation in scleroderma has been used as evidence of adrenal dysfunction.[2] Brooks[22] mentions the fact that the thymus and gonads influence calcium metabolism and in that way may affect calcinosis in scleroderma. Roederer[36] was able to produce marked improvement in scleroderma in an 18 year old female by using pluriglandular organotherapy. More general discussions on the subject of endocrine dysfunction in scleroderma can be found in the papers by Boardman,[37] Foerster,[30] Roques,[5] Howell[1] and Castle.[6] There is not sufficient evidence to support seriously any one or group of these endocrine glands as the cause for scleroderma. The results reported might be only coincidental, since remissions and exacerbations are so characteristic of the disease.

THE NERVOUS SYSTEM

Sympathetic Nervous System: The frequently symmetrical distribution of the lesions of scleroderma and sclerodactylia, especially in the upper extremities, prompted a suspicion many years ago that the nervous system might be responsible for the disease.[2, 3, 13, 37, 38] Again, the frequent association of scleroderma with Raynaud's disease, which has been considered by some to be due, at least in part, to vasomotor phenomena, is used as evidence for sympathetic dysfunction in scleroderma.[2, 3, 13, 39, 40] The beneficial results obtained by some investigators following interruption of the sympathetic pathways apparently lends further support to the idea that malfunction of the sympathetic nervous system contributes to the cause of scleroderma.[13, 41, 42] Acetyl-beta-methylcholine chloride (mecholyl) iontophoresis has been used in scleroderma[43] with the idea of relieving vasospasm, which is said to be abnormally increased. Certain abnormal histologic changes have been described for the sympathetic nervous system, such as sympathetic ganglia, dorsal root nerves, anterior horn of the cord and the like.[3] The studies of Brown et al.[10] on the effect of temperature on the arterioles and capillaries of the fingers in scleroderma suggest increased vasospasm. From their studies they felt that sympathetic impulses should be released in an attempt to curtail this vasospasm. Again, the high incidence of scleroderma in patients with neuroses suggests a sympathetic relationship.[1, 2, 8] As early as 1896, Dercum[9] pointed out the vagueness of the term "vasomotor neurosis." As mentioned by Howell,[1] the absence of frequent clinical manifestations of involvement of thoracic and abdominal sympathetics in scleroderma is somewhat difficult to understand in a disease that is due to sympathetic dysfunction. It is known, however, that some of the vertebral ganglia send all, or nearly all, their fibers to the body wall or limbs, and that some send all their fibers to the viscera. This may explain these observations. The absence of clinical manifestations of thoracic and abdominal sympathetic dysfunction does not necessarily mean that such changes do not occur, for as shown by Longcope[7] many patients with scleroderma have histologic changes in many of the visceral organs.

A more thorough discussion of this subject will be presented under Abnormal Physiology.

Central Nervous System: Histologic changes in the central nervous system and the spinal nerves have been described.[3, 8, 44, 45] Murray-Will,[46] in a study of the literature, found that the spinal fluid at times showed evidence of changes, such as an increase in the number of lymphocytes and an increase in albumin. Guillain et al.[47] found a non-luetic colloidal gold curve, a negative Wassermann, and an increase in the number of lymphocytes and in the amount of albumin in the spinal fluid of a patient with scleroderma. The "rheumatic" or neuralgic pain, itching, tingling, numbness and the like, so frequently observed at the onset of scleroderma, suggest a neurogenic aspect of the disease. This, however,

might be only a manifestation of neurologic effects of some other primary factor. Howell[1] was able to find a report of degenerative changes in the cells of the intermedio-lateral tract. Incidentally, sympathetic fibers arise from these same cells. Atrophic degeneration of the motor cells of the anterior horn was also noted. The patient had atrophy of the muscles previously innervated by these anterior cells. In another patient with sclerodermic hemiatrophy lesions were found in the anterior horn. Lesions around the ependyma have also been described. The frequent association of scleroderma and hemiatrophy suggests a nervous factor in the disease.[39] There is not sufficent evidence at present to relate scleroderma etiologically to lesions in the central nervous system.

VASCULAR DISTURBANCES

Most of the evidence favoring a vascular disturbance as an etiologic basis for scleroderma is founded upon observations of vascular physiology in involved areas. This will be discussed more thoroughly later on. However, a few observations which are of etiologic significance will be discussed at present.

It is a well known fact that scleroderma frequently follows Raynaud's disease, a condition with known vascular disturbances.[24, 48, 49] The frequent association of these two conditions suggested a vascular basis for the former disease as well. Furthermore, histologic studies of small arteries in sclerodermatous skin usually show an endarteritis with varying degrees of intimal proliferation and perivascular, as well as diffuse, infiltration of lymphocytes.[1, 2, 3, 10, 44, 50] Studies on the capillaries of the nail bed showed definite anatomic and functional disturbances, such as decrease in the number, size, shape, tone, and circulation rate of blood through these vessels.[10, 51] Lewis and Landis[50] have found experimental evidence of decreased arterial flow and increased arterial spasm in the type of scleroderma that follows Raynaud's phenomena. They have shown rather definitely that the factors responsible for this reduced flow are purely local phenomena and are not related to nervous impulses coursing through the central nervous system and down main nerve trunks. They were unable to find any evidence of abnormal sympathetic tone. The distribution of the lesions is also compatible with primary vascular disturbances, as well as with nervous disturbances. The histamine wheal test is definitely abnormal in scleroderma and is only slightly changed by sympathetic operation.[42, 52] Evidence for the above statement will be discussed below under Abnormal Physiology.

If abnormal vascular functions are responsible for scleroderma, the primary factor or factors responsible for the vascular pathology remain to be found.

MISCELLANEOUS CAUSES

Among the many causes advanced to explain the etiology of scleroderma, chronic arsenic poisoning, syphilis, nervous shock, trauma, exposure, infections of various kinds (measles, tonsillitis, scarlet fever, diphtheria, pneumonia, and influenza), and pregnancy are among the most common.[2, 37, 39, 46, 53-57] Most of these factors have not been proved to cause scleroderma, or even to be etiologically related. Some are probably no more than coincidental factors. Boardman[37] has written an extensive review on the etiology and treatment of scleroderma, and is recommended for more thorough discussion of these subjects. His bibliography is quite complete.

Moench[53] reported a patient with arsenic poisoning, scleroderma and neuritis. Arsenic was found to be present in high quantities in the urine. The use of sodium thiosulfate increased the symptoms of neuritis as well as the elimination of arsenic. Ayres[58] reported three patients with arsenic in the urine and a history of contact with arsenic who also had sclerodermatous changes. No other abnormal findings were detected. He pointed out the fact that 43 per cent of 48 specimens of urine selected at random contained arsenic, and suggested that to be of any significance arsenic would have to be present in the urine of sclerodermatous patients in a much higher concentration. This point still remains unset-

tled. In individual cases, scleroderma and chronic arsenic poisoning possess many symptoms in common, such as neuritic manifestations, pigmentation, cutaneous alterations of various kinds, and loss of weight. Again, many of these symptoms may be lacking in cases of well advanced scleroderma. Brooke and Roberts[54] and Barendt[55] described skin changes in many individuals following arsenic poisoning developed from drinking beer containing large amounts of arsenic. These skin changes, however, appeared to be more like those described in arsenical dermatitis than those of scleroderma, although there were many similar manifestations, such as erythema, edema, dirty-brown to coppery discoloration, hyperkeratosis, desquamation and paresthesias. The relationship of arsenic poisoning to scleroderma needs further study, and at present one cannot conclude that the latter condition is a manifestation of the former.

Syphilis has been blamed at some time or another for most of the diseases which possess an obscure etiology and, as one would expect, it has been said to cause scleroderma.[2, 46] Murray-Will[46] reported a patient with scleroderma in whom a positive blood Wassermann was found, and who failed to improve under the ordinary methods of scleroderma therapy, but improved following antiluetic therapy. He reviewed the literature with this observation in mind and found further evidence to support the association of syphilis with morphea, Raynaud's disease and sclerodactylia. However, most of the patients with scleroderma reported in the literature have no evidence of syphilis, and at present there are not sufficient data to indicate more than a coincidental relationship of syphilis to scleroderma.

The generalized nature of the disease, the constitutional manifestations, such as fever, malaise, weakness, and metabolic changes, which often usher in diffuse scleroderma, suggest a toxic or infectious basis for the disease. Leprosy, once thought to have a possible relationship with scleroderma, has been shown to be unrelated, although scleroderma-like lesions have been described in leprosy.[59]

The other factors advanced to explain the cause of scleroderma lack sufficient proof to justify their being considered more than aggravating factors. It is well to eliminate such influences when treating a patient with scleroderma as they may have some detrimental effect, if not on the sclerodermatous processes per se, perhaps constitutionally.

ABNORMAL PHYSIOLOGY

Studies of functional changes in parts affected with scleroderma are wanting. In recent years definite promising advances have been made in that phase of the disease. Before attempting to discuss these observations, it is necessary, as will be realized later, to present a classification of scleroderma, a number of which have been advanced. The one presented by O'Leary and Nomland[2] is as good as any. It is:

1. The diffuse or generalized type.
2. The localized type:
   a. Morphea.
   b. Scleroderma in bands.
   c. Guttate scleroderma.

The diffuse or generalized type frequently follows Raynaud's phenomena, but not necessarily.[2, 49, 51] It has been shown[50, 51] to be closely related to this condition as far as the vascular characteristics are concerned. Again, this type has been the one most frequently studied. When the fingers are involved by the sclerodermatous changes the condition is referred to as sclerodactylia. There are various opinions as to whether or not morphea and diffuse scleroderma are related and should be classified together.[6] However, the majority of evidence favors a relationship and a grouping together.[37]

Brown and O'Leary[51] studied the capillaries of the finger nail bed in patients with scleroderma and found definite abnormal changes which were similar in many respects to those found in patients with Raynaud's disease alone.[60] They studied five patients of three groups: (1) The diffuse, primary, progressive scleroderma; (2) the borderline, or secondary, vasomotor type, and (3) the localized form, or morphea. The first two groups are of the diffuse or gen-

eralized type shown in the classification above. In the first group the most striking finding was the presence of large loops or so-called giant capillaries. The capillaries were markedly reduced in number, there being only one-third the normal number. The decrease in number of loops "seemed to stand in direct relationship to the degree of trophic disturbance." The diameters of the arterial and venous portions of the loops were increased and were at times large enough to obliterate the space between the two limbs. Temperature changes and mechanical irritation failed to produce detectable changes in the caliber of these vessels. The capillary outline was "usually frayed and indistinct, giving the loops a feathery appearance." This was due to the decreased transparency of the skin as a result of the collagenous hypertrophy. In the vasomotor type, or second group, the capillaries were found to present an entirely different picture, sufficiently different to distinguish this group from the first. The capillaries resemble those found in Raynaud's disease.[60] They were decreased in number but preserved the usual capillary pattern; large giant loops were occasionally seen, while the large shapeless loops described in the first group were not seen. Decreased tonus with dilatation of the arterial and venous portions was noted. The loops were slightly longer than normal. The flow through these capillaries was very sluggish and many loops showed stasis with cyanosis. As noted in Raynaud's disease[60] during the pallor or the syncope phase, contracted, partially filled loops were present and many were invisible. "In the asphyxia phase, clumps of cells were seen to enter the loops slowly from the arterial and venous sides; then complete stasis, increasing cyanosis and dilatation of the loops developed." With reestablishment of flow and the presence of oxyhemoglobin the rubor phase developed.

The authors said little about the changes in morphea. Brown and O'Leary were unable to state whether the capillary changes were primary or secondary causes of the skin changes. They likewise were unable to evaluate the effect of the fibrotic change of the corium on the vessels. They felt that the stasis and agglutination phenomena in the capillaries increased the capillary permeability, which in turn was responsible for edema. Pautrier and Ullmo[61] found irregularities in the capillaries and a marked decrease in the number as well as in the diameters of the capillaries in scleroderma. Their findings as a whole agreed with those of Brown and O'Leary. These findings indicate a definite reduction in the volume of blood flow to the part.

Lewis and Landis[50] and Lewis and Kerr[48] reported a group of very interesting studies on Raynaud's disease with and Raynaud's disease without scleroderma, respectively, which tended to show that in Raynaud's disease with or without scleroderma there were local organic and functional disturbances in the small peripheral arteries. These disturbances were apparently not influenced by the presence or absence of scleroderma. They noted that there was an optimum temperature range (10°-22°) at which asphyxia and cyanosis could be produced in these patients.[48] Lewis and Landis[50] found that by cooling the whole hand in water the characteristic asphyxial change was produced from the finger tip proximally; cooling of the finger tip alone produced no such change. Again, heating of the finger tip after the hand had been previously made cyanotic produced no change, while submerging the hand and fingers except the tips promptly relieved the spasm, and the whole hand, tips and all, became pink. They also noted that if the ulnar side of the hand were immersed in cool water that portion of the hand and the submerged fingers became cyanotic while the fingers and part of the hand above water remained pink. If a patient had cyanosis of both hands from cooling, and the wrist, palm and thumb of the left hand were submerged in warm water (35° C.), and the tips of the fingers were left protruding out of the water, the whole submerged portion and protruding finger tips became pink. Now, if at the same time only the tips of the fingers of the equally cyanotic right hand were submerged in water at 35° C., no change occurred, not even for the submerged tips. Lewis and Landis further noted that

ulner nerve anesthesia did not alter the experiments in any part of the hand, not even the anesthetic fifth finger. All of these findings were essentially the same in the hands and finger tips of patients with Raynaud's disease alone.[48] The authors state that "the evidence that has now been brought together is sufficient to show beyond any reasonable doubt that in the type of case studied, in which circulatory changes in the finger are combined not only with local but also with distant scleroderma, the circulatory disturbances arise, as they do in the common type of Raynaud's malady . . :, from a fault in the vessels themselves." These studies showed the disturbance to be local, that is in the digital arteries. Their studies further showed that patients with mild organic change were not very apt to develop permanent spasm, while those with more severe organic changes experienced more permanent spasm, the spasm being reinforced by local, structural changes.

In discussing the morbid changes, Lewis and Landis[50] state (quoting them almost verbatim) that in the case of the more severe forms of Raynaud's disease, those in which evidence of structural disease of the arteries is obtainable, tests show that closure of the vessels is brought about easily and over an unusually wide range of temperature. It is quite possible, if it is not probable, that the impression of unusual susceptibility to spasm in the digital vessels of these patients is in part due to the vessels' closing prematurely owing to the thickness of their intimal lining. (The reviewer might add that with intimal proliferation and narrowing of the lumen of the vessel, the surface area of the intima is decreased, and without change in blood pressure the intravascular force exerted on the wall of the vessel is reduced; therefore, the same normal amount of nervous stimulus and force of smooth muscle contraction of the media will be more effective in overcoming the intravascular force and thereby more effectively produce constriction of the vessel. This probably would result in a greater degree of closure than would be expected under ordinary circumstances. Again, with the irregularities in location and variations in degrees of intimal thickening, one wonders to what extent the Bernoulli phenomenon might mechanically affect the behavior of these diseased vessels and the volume of blood flow through them.) Just in so far as this factor enters into play, Raynaud's disease in its severer forms must be regarded as a structural rather than a spasmodic arterial disease. It is not suggested, however, that it is right to regard the disease as ever purely structural, but only that structure change may play some part in bringing the vessels, that is the seat of spasm, to a state of complete obliteration. These patients behave in a very different way from patients with thrombotic disease of the digital vessels occurring from time to time in thrombo-angiitis obliterans and senile arteritis, although the disease in such vessels, displayed by the authors'[50] inability to dilate them by heat, is as advanced or more advanced than that in scleroderma. It is not the habit of fingers so affected to become fully livid, white or numb owing to prolonged arrest of the circulation, on exposure to cold atmospheres, and spasm cannot be induced, as it can in Raynaud's malady, in different parts of the digital vessel at will by suitably confining the action of cold to different regions.

Thus, while the severer, like the milder, case of Raynaud's disease should continue to be regarded as essentially a spasmodic affection, yet it is to be recognized that structural change may contribute to the circulatory defect. It contributes by failing to permit the vessel to open to its fullest when heated; it contributes, as previously stated, by bringing the vessel to premature closure when the part is cooled.

Prinzmetal[40] studied the effect of the tight atrophic skin on the circulation in the digits of subjects suffering from sclerodactylia. He found that the circulation to the digits was further impaired by the tight inelastic skin and subcutaneous tissues which contract down around the underlying blood vessels further diminishing the blood flow to the part. His studies suggest that the atrophy of the terminal phalanx in sclerodactylia is probably due to the pressure by the tight skin. He found that sympathec-

tomy raised the temperature of the fingers of normal subjects and subjects without sclerodactylia, but produced only a slight or no rise in temperature in patients with sclerodactylia. The skin prevents the vessels from dilating once sympathetic tone is released. Relaxation of the skin in one patient resulted in improvement of the circulation. The volume of pulsations in the fingers was smaller in subjects with sclerodactylia than in those without. Vasodilator impulses by the Landis heat test produced little change in temperature. Histamine also failed to produce a rise in temperature in these patients. By placing a tight finger cot on normal fingers Prinzmetal was able more or less to reproduce the conditions encountered in sclerodactylia. He suggests that these patients need a procedure that would relax this tight skin in order to improve satisfactorily with treatment.

Turner, Burch and Sodeman[62] found the pulse volume and total blood volume in the finger tip affected with Raynaud's disease to be definitely reduced. Johnson and Hedges[63] also found a reduced volume of pulsations in the finger tip involved with scleroderma. Sodeman and Burch[64] found the tissue pressure to be markedly elevated in subjects suffering from scleroderma. These latter studies gave direct evidence for a mechanism, that is mechanical pressure, whereby vascular changes may be secondary to the sclerodermatous process, and as well supported Prinzmetal's contention that this contributes in part to the disturbed vascular function. These observers[64] state that "with tissue pressures of the magnitude which we have found, which have exceeded the normal venular and capillary pressures, one would expect a collapse of these vessels. Permanent collapse would be prevented by a building up of the venular and capillary pressures from the arteriolar side, provided no collateral circulation were possible.

"It is conceivable that in a localized area, if the process were severe, and if a collateral circulation were adequate, a rise in tissue pressure, exceeding the venous pressure of the adjoining tissues, could result in localized ischemia and atrophy. Proximal to the involved areas in which the tissue pressure exceeds the normal venous pressure, the venous pressure would fall to its usual normal value. This is exemplified by the conditions existing when a pneumatic cuff about the arm is inflated to a value exceeding the normal venous pressure and less than diastolic blood pressure. In such an instance the venous pressure distal to the cuff will build up and equal the cuff pressure to reestablish the circulation, while the venous pressure proximal to the cuff is unaffected. As a routine measure, venous pressure measurements are difficult in these patients because of the character of the skin and the small size of the veins in the involved areas. Fortunately, however, a small vein was accidentally entered while taking the tissue pressure in the dorsum of the forearm in one of our patients. The venous pressure in this vein at heart level was 228 mm. of water, while the pressure at heart level in the uninvolved antecubital space was 86 mm. of water." These same observers also noted that with the development of edema in sclerodermatous areas the tissue pressure increased further and as the edema subsided the tissue pressure fell.[64, 65] The hardness of the skin and subcutaneous tissues in scleroderma was further determined objectively by Burch and Sodeman[66] with the use of a specially constructed tonometer for the finger tip which makes it possible to follow changes in that region. With the use of a "distensiometer" Sodeman and Burch[67] were able to express in a quantitative fashion the tightness or "stretchability" of the human skin. The skin of scleroderma was found to be, relatively speaking, extremely non-distensible. This lends further support to the discussions above. These quantitative apparatus—tissue pressure apparatus, tonometer, and distensiometer—are steps toward the development of more quantitative means for measuring changes in vascular states. These methods can be applied in clinics and laboratories and serve a useful purpose by more accurately evaluating changes as well as methods of treatment in

scleroderma than in the past, when subjective methods were employed almost entirely.

The studies of King,[52] and Caldwell and Mayo[42] in scleroderma with the histamine wheal, in which the triple response of Lewis was found to be absent, suggest impairment of blood flow due to disturbances in the small arterial vessels.

It appears, therefore, that in scleroderma the vascular pathology, endarteritis and intimal thickening, of the small arteries, decrease in the number of capillaries, the contracting fibrous subcutaneous tissues, and the possibly increased sympathetic tone, all tend to impair the circulation to the affected part.

The temperature of fingers affected with sclerodactylia is lower than that of normal or unaffected fingers and tends to remain below room temperature.[10, 40] The hands of these patients are cold and clammy and have a cadaveric appearance. Brown, O'Leary and Adson,[10] using the Stewart and Kegerreis calorimeter, found the heat loss in these involved areas to be markedly reduced below normal, suggesting a definite reduction in blood flow. They found no significant differences for the various forms of scleroderma. These same observers applied Brown's method for determining the "vasomotor index"[68] to patients with scleroderma. They found that there was less evidence of vasomotor spasm in patients with scleroderma without associated Raynaud's disease than in those with associated Raynaud's disease. They attribute this to the additional factor of occlusive disease of the smaller arterioles. They found that by lowering the room temperature they could produce complete cessation of blood flow in the capillaries, which they attribute to closure of the arterioles due to an abnormal degree of spasm.

In Raynaud's disease with scleroderma the syncope, asphyxia, small pulse volume in the involved area, and other vascular phenomena, as well as histologic changes in the various centers of the sympathetic nervous system, pathways and the like, suggest that in scleroderma there may be an associated sympathetic disturbance. As discussed previously, most of these findings can be explained, however, on the basis of localized organic vascular pathology. Adson, O'Leary and Brown[41] and Leriche and his associates[13] have found that resection of sympathetic ganglia and trunks, in an attempt to relieve an excessive amount of vasospasm, has benefited patients suffering with scleroderma. Most of the evidence for increased amount of sympathetic tone, independent of localized vascular disease in scleroderma, is based upon the relationship of scleroderma to Raynaud's disease[2] and the beneficial results reported following the interruption of sympathetic nervous pathways. As stated by Lewis and Kerr,[48] these operations remove the vasoconstrictor tone and the results obtained following such operations are just what would be expected when normal vasoconstrictor tone is removed. They can see no reason to conclude that such observations constitute evidence of abnormal vasoconstrictor tone. The studies of Lewis and Kerr[48] and Lewis and Landis,[50] previously discussed, tend to show that the abnormal vascular phenomena present in Raynaud's disease with or without scleroderma are the result of local morbid arterial changes and are independent of sympathetic tone.

Certain metabolic, including muscle metabolism,[69] and chemical changes have been studied sporadically but need not be discussed here because their significance is not known. Some of these pathologic physiologic aspects of calcium and phosphorus metabolism and the endocrines have been discussed and need not be repeated here.

SUMMARY AND CONCLUSIONS

Many factors have been said to cause scleroderma. Of these, hyperfunction of the parathyroids, hypofunction of the thyroid, increased sympathetic tone, and local peripheral vascular disease have been most seriously considered. There is not sufficient evidence at present properly to evaluate the relative significance of these factors.

Studies of the disturbed physiology have showed evidence of abnormal calcium and phosphorus metabolism, decreased blood supply to the involved area and also increased vasospasm, which is most probably localized to the small peripheral arteries. The decreased blood flow to the involved part is due, at least in part, to intimal thickening of the small arteries, decrease in number and abnormal function of the capillaries, and to the increased local vasospasm.

A number of methods for quantitatively following the progress of scleroderma with or without treatment were discussed. The value of such quantitative measurements made at frequent intervals in an attempt to evaluate therapeutic procedures was mentioned.

REFERENCES

1. Howell, A.: Diffuse scleroderma, Practitioner, 93:604, 1914.
2. O'Leary, P. A. and Nomland, R.: A clinical study of one hundred and three cases of scleroderma, Am. J. Med. Sci., 180:95, 1930.
3. Rake, G.: On the pathology and pathogenesis of scleroderma, Bull. Johns Hopkins Hosp., 48:212, 1931.
4. Osler, W.: On diffuse scleroderma, with special reference to diagnosis and to the use of thyroid gland extract, J. Cutan. & Genito-Urin. Dis., 16:49 and 127, 1898.
5. Roques, E.: Le traitment opothérapique de la sclerodermie, Ann. de dermat. et de syph., 11:383, 1910.
6. Castle, W. F.: The endocrine causation of scleroderma, including morphea, British J. Dermat. & Syph., 35:255 and 303, 1923.
7. Longcope, W. T.: Hypoglycemia in scleroderma, J. A. M. A., 90:1, 1928.
8. Oliver, E. L. and Lerman, J.: Scleroderma treated with injection of posterior pituitary extract, Arch. Dermat. & Syph., 34:469, 1936.
9. Dercum, F. X.: Scleroderma, J. Nerv. & Ment. Dis., 21:431, 1896.
10. Brown, G. E., O'Leary, P. A. and Adson, A. W.: Diagnostic and physiologic studies in certain forms of scleroderma, Ann. Int. Med., 4:531, 1930.
11. Aub, J. C., Bauer, W., Heath, C. and Ropes, M.: Studies of calcium and phosphorus metabolism. III. The effects of the thyroid hormone and thyroid disease, J. Clin. Investigation, 7:97, 1929.
12. Cornbleet, T. Struck, H. C.: Calcium metabolism in scleroderma, Arch. Dermat. & Syph., 35:188, 1937.
13. Leriche, R., Jung, A. and DeBakey, M. M.: The surgical treatment of scleroderma. Rationale of sympathectomy and parathyroidectomy, Surgery, 1:6, 1937.
14. Bernheim, A. R. and Garlock, J. H.: Parathyroidectomy for Raynaud's disease and scleroderma, Ann. Surg., 101:1012, 1935.
15. Leriche, R. and Jung, A.: Recherche sur la nature de la sclérodermie. Les traductions tissulaires de l'hyperparathyroïdism dans la sclérodermie, Presse méd., 43:1361, 1935.
16. Garlock, J. H.: Parathyroidectomy for Raynaud's disease and scleroderma, Surg. Clin. North America, 16:771, 1936.
17. Selye, H.: A condition simulating human scleroderma in rats injected with parathyroid hormone, J. A. M. A., 99:108, 1932.
18. Shelling, D. H., Asher, D. E. and Jackson, D. A.: Calcium and phosphorus studies. VII. The effects of variations in dosage of parathormone and of calcium and phosphorus in the diet on the concentration of calcium and inorganic phosphorus in the serum and on the histology and chemical composition of the bones of rats, Bull. Johns Hopkins Hosp., 53:348, 1933.
19. Leriche, R., Jung, A. and Sureyya, C.: La peau dans l'hyperparathyroïdism experimental, étude de la sclérodermie experimentale, Presse méd., 43:777, 1935.
20. Hueper, W.: Metastatic calcifications in the organs of dogs after injection of parathyroid extract, Arch. Path. & Lab. Med., 3:14, 1927.
21. Durham, R. H.: A case of scleroderma with extensive subcutaneous periarticular and vascular calcification, Ann. Clin. Med., 5:679, 1926.
22. Brooks, W. D. W.: Calcinosis, Quart. J. Med., 3:293, 1934.
23. Gordon, H.: Diffuse scleroderma, with case report and autopsy findings, Ann. Int. Med., 2:1309, 1929.
24. Pautrier, L. M.: Sclérodermies, chéloïdes et calcémie, Presse méd., 41:345, 1933.
25. Gitlow, S. and Steiner, S.: Scleroderma with special reference to the blood chemistry, Arch. Dermat. & Syph., 9:549, 1924.
26. Durham, R. H.: Scleroderma and calcinosis, Arch. Int. Med., 42:467, 1928.
27. Mamon, H.: Sclérodermie et parathyroïdes, Gaz. d. Hôp., 105:375, 1932.
28. Kennedy, R. L. J.: Calcinosis and scleroderma in a child treated by ketogenic diet, Med. Clin. North America, 12:1655, 1929.
29. Leriche, R. and Jung, A.: Le chlorure d'ammonium dans la thérapeutique de la sclérodermie, Presse méd., 41:1041, 1933.
30. Foerster, O. H.: The relation of internal secretions to cutaneous disease, J. Cutan. Dis., 34:1 1916.
31. Fox, G. H.: Morphea, J. Cutan. Dis., 35:256, 1917.
32. Winfield, J. M.: A case of scleroderma improved under the use of desiccated suprarenal gland, J. Cutan. Dis., 22:586, 1904.
33. Millard, M.: Sclérodermie améliorée par l'arrhénal, Bull. et mém. Soc. méd. d. hôp. d. Paris, 22:163, 1905.
34. Jeanselme and Buinier: Sclérodermie en plaques avec dyschromie pigmentaire symétrique, Bull. Soc. franç. de dermat. et syph., 36:704, 1926.
35. Wahl, E. F.: Scleroderma. Insulin therapy, J. Med. Assn. Georgia, 19:285, 1930.
36. Roederer, M. J.: Sclérodermie en plaques améliorée par l'opothérapie pluriglandulaire, Bull. Soc. franç. de dermat. et syph., 36:231, 1926.
37. Boardman, W. P.: Scleroderma, with special reference to its etiology and treatment, Arch. Dermat. & Syph., 19:901, 1929.
38. Fagge, C. H.: Keloid, scleriasis, morphea, and some allied affections, Guy's Hosp. Repts., Third series, 13:255, 1868.
39. McCrackin, R. H.: Scleroderma. Report of a case with scleroderma and hemiatrophy, J. Pediat., 9:173, 1936.
40. Prinzmetal, M.: Studies of the mechanism of circulatory insufficiency in Raynaud's disease in association with sclerodactylia, Arch. Int. Med., 58:309, 1936.
41. Adson, A. W., O'Leary, P. A. and Brown, G. E.: Surgical treatment of vasospastic types of scleroderma by resection of sympathetic ganglia and trunks, Ann. Int. Med., 4:555, 1930.
42. Caldwell, J. M., Jr. and Mayo, J. G.: Cutaneous reactions to histamine. Reactions in occlusive and spastic vascular disease and in chronic infectious arthritis, Arch. Int. Med., 47:403, 1931.
43. Duryee, A. W. and Wright, I. S.: The treatment of scleroderma by means of acetyl beta methyl choline chloride (mecholyl) iontophoresis, Am. Heart J., 14:603, 1937.
44. Robinson, A. S.: Report of a case of scleroderma of the skin, Am. J. Med. Sci., 154:657, 1917.

45. Williams, C. M.: A case of diffuse scleroderma presenting unusual features, Arch. Dermat. & Syph., 9:187, 1924.

46. Murray-Will, E.: Scleroderma and syphilis, British J. Dermat. & Syph., 39:201, 1927.

47. Guillian, G., Alajouanine, T. and Marquézy, R.: Sclérodermie progressive avec cataracte double précoce chez un infantile, Bull et mém. Soc. méd. d. hôp. de Paris, 47:1489, 1923.

48. Lewis, T. and Kerr, W.: Experiments relating to the peripheral mechanism involved in spasmodic arrest of the circulation in the fingers, a variety of Raynaud's disease, Heart, 15:7, 1929-31.

49. Lewis, T. and Pickering, G. W.: Observations upon maladies in which the blood supply to digits ceases intermittently or permanently, and upon bilateral gangrene of digits; observations relevant to so-called "Raynaud's disease", Clin. Sci., 1:327, 1933-34.

50. Lewis, T. and Landis, E. M.: Further observations upon a variety of Raynaud's disease; with special reference to arteriolar defects and to scleroderma, Heart, 15:330, 1929-31.

51. Brown, G. E. and O'Leary, P. A.: Skin capillaries in scleroderma, Arch. Int. Med., 36:73, 1925.

52. King, A. D.: Local responses to histamine in normal skin, in dermatophytosis, alopecia areata and in scleroderma, Arch. Dermat. & Syph., 25:840, 1932.

53. Moench, L. M.: A case of chronic arsenic poisoning in a child associated with profound anemia and scleroderma, New York State J. Med., 36:1029, 1936.

54. Brooke, H. G. and Roberts, L.: The action of arsenic on the skin as observed in the recent epidemic of arsenical beer poisoning. British J. Dermat. & Syph., 13:121, 1901.

55. Barendt, F. H.: The skin lesions due to the presence of arsenic in beer, British J. Dermat. & Syph., 13:148, 1901.

56. Putnam, J. J.: The frequency with which arsenic is found in the urine; with notes on some few cases of poisoning from wall-paper and fabrics, Boston M. & S. J., 122:421, 1890.

57. Gaskill: Scleroderma, J. Cutan. Dis., 34:465, 1916.

58. Ayres, S., Jr.: Scleroderma as a possible manifestation of chronic arsenic poisoning, Arch. Dermat. & Syph., 2:747, 1920.

59. Seale, E. R.: Endocrine aspects of scleroderma. Report of a case with glandular dysfunction, Southern M. J., 22:885, 1929.

60. Brown, G. E.: Skin capillaries in Raynaud's disease, Arch. Int. Med., 35:56, 1925.

61. Pautrier, M. L.-M. and Ullmo, A.: Note sur la capillaroscopie de la dermatite chronique atrophiante et de la sclérodermie, Bull. Soc. franç. de dermat. et syph., 36:776, 1929.

62. Turner, R. H., Burch, G. E. and Sodeman, W. A.: Studies in the physiology of blood vessels in man. III. Some effects of raising and lowering the arm upon the pulse volume and blood volume of the human finger tip in health and in certain diseases of the blood vessels, J. Clin. Investigation, 16:789, 1937.

63. Johnson, C. A. and Hedges, R. N.: Studies on peripheral vascular phenomena. IV. Finger volume changes in a patient showing Raynaud's phenomenon, Surg., Gynec. & Obstet., 60:1077, 1935.

64. Sodeman, W. A. and Burch, G. E.: Tissue pressure: An objective method of following skin changes in scleroderma, Am. Heart J., 17:21, 1939.

65. Sodeman, W. A. and Burch, G. E.: The tissue pressure in subcutaneous edema, Am. J. Med. Sci., 194:846, 1937.

66. Burch, G. E. and Sodeman, W. A.: A tonometer for measurement of tissue turgor in the human finger tip, Proc. Soc. Exper. Biol. & Med., 39:125, 1938.

67. Sodeman, W. A. and Burch, G. E.: A direct method for the estimation of skin distensibility with its application to the study of vascular states, J. Clin. Investigation, 17:785, 1938.

68. Brown, G. E.: The treatment of peripheral vascular disturbances of the extremities, J. A. M. A., 87:379, 1926.

69. Epstein, E. and Ayres, S., Jr.: Creatinuria in scleroderma, J. Lab. & Clin. Med., 22:697, 1937.

SURGICAL CONSIDERATIONS

ALTON OCHSNER, M. D.†

AND

MICHAEL DEBAKEY, M. D.†

NEW ORLEANS

The surgical treatment of scleroderma is based upon a rational consideration of certain clinical and experimental observations which seem to have pathogenic significance. In previous publications (with Leriche and Jung[1, 2]) these factors which form the basis of parathyroidectomy and sympathectomy were reviewed, and the results obtained following the application of these surgical procedures in 26 cases of scleroderma were presented. At that time it was stated that improvement to a greater or lesser degree followed parathyroidectomy in over 90 per cent and sympathectomy in approximately two-thirds of the cases. However, the methods of determining such improvement admittedly were not accurate as they depended upon subjective and relatively gross objective means. With the development of more accurate methods of study devised by Burch and Sodeman,[3, 4, 5] it has been possible to determine with considerable accuracy the presence or absence as well as the degree of improvement following therapy. Accordingly, four patients operated upon by us have been studied in an attempt to evaluate better the results of the surgical procedures.

RATIONALE OF SURGICAL PROCEDURES

Whereas a detailed discussion of the clinical and experimental factors which form the basis of parathyroidectomy and sympathectomy in the treatment of scleroderma is beyond the scope of this presentation, it is considered desirable to review briefly the rationale of these surgical proce-

†From the Department of Surgery, School of Medicine, Tulane University, New Orleans, La.

dures. Parathyroidectomy is based upon certain clinical and experimental observations which seem to indicate a definite physiologic disturbance in the calcium metabolism suggesting a hyperfunction of the parathyroids. That hyperparathyroidism may play a prominent role in the pathogenesis of scleroderma has been expressed by numerous observers. Amongst the first to direct attention to the pathogenic significance of parathyroid dysfunction in this disease are Leriche and his co-workers[1, 2, 6-10] and Weissenbach and his collaborators.[11-14]

HYPERPARATHYROIDISM

Leriche and Jung[9-10] are convinced that scleroderma is the result of an abnormal increase of calcium in the skin consequent to a skeletal "osteolysis" due to hyperparathyroidism. It is their conception that the endocrine dysfunction, i. e., chronic hyperparathyroidism, is the primary disturbance, the bone changes or osteolysis the intermediate, and the cutaneous process the end-result. Weissenbach and his collaborators[11-14] have emphasized further the parathyroid dysfunction in scleroderma. Bernheim and Garlock[15, 16] have expressed the belief that disturbance in calcium metabolism associated with hyperplasia and hypertrophy of the parathyroids is responsible for the vasospastic as well as the cutaneous manifestations occurring in scleroderma. The parathyroid tissue removed at operation in six cases of scleroderma showed hypertrophy in every instance and evidence of acute parathyroiditis in one according to these authors.[15] Similar histopathologic changes have been observed by others.[17, 18] Of interest in this regard is the report by Aisenberg[19] of a case of scleroderma following malaria in which the author expresses the opinion that the disease resulted from parathyroid injury by the malarial parasite.

In true hyperparathyroidism there usually occurs hypercalcemia, hypophosphatemia, and a negative calcium balance. The results of such studies in scleroderma have been somewhat confusing. Because these characteristic findings have not been constantly present in scleroderma, many observers have urged that a hyperparathyroidism cannot be present. That hypercalcemia may occur in scleroderma has been demonstrated by numerous investigators.[20-29] In one of our cases there was a marked hypercalcemia and following parathyroidectomy the blood calcium returned to and remained normal. On the other hand, a number of observers[18, 28, 30, 31] have reported normal or even subnormal blood calcium findings in patients with scleroderma. However, as previously stated by Leriche, Jung and DeBakey[1] such findings do not necessarily "depreciate the significance of hypercalcermia in scleroderma." It has been shown that in experimental chronic hyperparathyroidism the blood calcium is at first increased but within several months drops to and remains normal.[1, 32] As scleroderma should be considered a chronic parathyroid disturbance rather than an acute one, the blood calcium studies may not be significant. Similarly, corresponding findings are to be expected in the urinary calcium. Of particular interest in this regard is the authoritative opinion of Allbright, Aub, and Bauer[33] who state that, "Whereas hypercalciuria and hyperphosphaturia are two of the most constant metabolic abnormalities of hyperparathyroidism, from a practical clinical point of view, this evidence is extremely laborious to obtain, seldom necessary, and, in border-line cases often not helpful. Thus, hypercalciuria is dependent on the hypercalcemia. If the latter is at a border-line level, the former will be as well." A similar opinion is expressed by Bernheim and Garlock.[15]

Experimental support of the significance of hyperparathyroidism in scleroderma has been presented by a number of investigators. While studying the effect of intraperitoneal injections of parathyroid extract in young rats, Selye[34] observed cutaneous changes which closely resembled those occurring in scleroderma. Subsequently these findings were confirmed by others.[35, 36] Moreover, Leriche and his co-workers[36] found that the calcium content of the skin of these animals was increased two to three

times the normal. On the other hand, Hueper[37] was unable experimentally to reproduce these cutaneous changes. Curiously enough he employed dogs, whereas all the other investigators used rats. Clinically the calcium content of the skin in patients with scleroderma is definitely increased.[10] This has been demonstrated roentgenologically as well as by chemical analysis. In one of our cases, large deposits of calcium were observed roentgenologically in the skin over the hands, elbows, and axillae.

Because in classical examples of hyperparathyroidism skeletal decalcification is one of the characteristic manifestations, it may be argued that this should be observed in scleroderma if the condition is due to parathyroid disturbance. Durham[30] has contended that there is no evidence of generalized decalcification of bone in scleroderma. The decalcification most commonly observed occurs in the involved extremities and this may be attributed to disuse. On the other hand, diffuse decalcification, as revealed by roentgenograhic studies, has been observed by Leriche and his co-workers.[1, 9] However, as previously stated[1] some cases of scleroderma show no skeletal changes. This does not necessarily indicate absence of disturbed parathyroid function because as stated by Brody and Bellin,[38] the process of removal of calcium from the bones through the action of the parathyroids may be very slow, "so that if there is an adequate intake of calcium the amounts removed from the bones are replaced and little or no osteoporosis is observed."

PARATHYROIDECTOMY

On the basis of these clinical and experimental observations which indicated that in scleroderma there occurs a definite physiologic disturbance in the calcium metabolism suggesting parathyroid hyperfunction, parathyroidectomy was considered a rational therapeutic procedure. Accordingly Leriche,[8] in 1931, performed the first parathyroidectomy in a case of scleroderma. The operation was followed by a remarkable improvement. Subsequently others[14-16, 39-41] have made similar observations. Leriche, Jung, and DeBakey[1] in an analysis of 13 cases found that five (38.4 per cent) showed marked improvement, seven (53.8 per cent) showed moderate improvement, and only one (7.6 per cent) showed no improvement. Moreover, there were six cases observed from two to five years after operation. In all of these the improvement not only was maintained but continued. Two of these patients observed two and five years, respectively, after operation were apparently completely relieved of all previous manifestations. We have performed parathyroidectomy in combination with sympathectomy in three patients with scleroderma. These patients were carefully studied by means of the distensiometer, tonometer, and tissue pressure apparatus devised by Burch and Sodeman.[3, 5] Such objective methods permit accurate evaluations of any changes which may occur following the institution of therapy. The results of these studies in the three patients in whom parathyroidectomy was performed approximately one and a half years ago reveal only slight improvement in one, moderate improvement in the second, and marked improvement in the third.

SYMPATHECTOMY

Sympathectomy is the other surgical procedure which has been applied in the treatment of scleroderma. This is based upon certain clinical and experimental observations which seem to indicate that the sympathetic apparatus is of pathogenic significance in certain forms of scleroderma. It has long been observed that vasomotor disturbances closely simulating Raynaud's phenomena frequently precede the characteristic cutaneous manifestations of scleroderma.[42, 43, 44] In a clinical study of 103 cases, O'Leary and Nomland[43] found that this occurred in over one-third. The vascular and cutaneous manifestations observed in some of these cases are undoubtedly closely related.[42, 45] Microscopic studies of the capillaries in the nail bed made by Brown, O'Leary, and Adson[42, 46, 47] on patients with scleroderma and with Raynaud's alone revealed similar abnormal changes. These observations were also made by

Pautrier and Ullmo.[48] On the basis of their observations, Brown, O'Leary, and Adson are of the opinion that sympathetic hypertonia of the arterioles is a significant factor in the development of scleroderma. A similar opinion has been expressed by Thibierge and Weissenbach,[49] Follmann and Ballo.[50] The vasospastic element in scleroderma has been demonstrated further by oscillometric[51] as well as by plethysmographic studies.[52] The marked influence of environmental temperature, i. e., pronounced vasospasm on exposure to cold and vasodilation on exposure to heat, reflects the exaggerated tonus of the arterioles. On the other hand, Lewis and his co-workers[45, 53] are of the opinion that the pathologic vascular reactions observed in scleroderma are due to a local disturbance in the digital arteries. They believe that there exists an incomplete relaxation of the arteries between attacks of spasm and that premature closure of the vessels results from the structural changes in the arterial wall. These observers state that this does not depend upon sympathetic hypertonus. According to Prinzmetal[54] the ischemia present in these cases is due to the constricting effect on blood vessels by the tightness of the skin and not to sympathetic influence.

VASCULAR DISTURBANCES

That vascular disturbances play prominent roles in the development of scleroderma is shown further by histopathologic studies of the arterioles and capillaries. In detailed examinations of six patients, Matsui[55] found definite degenerative changes in the small cutaneous arteries as well as those of the lungs, kidneys, and endocrine glands. Similar findings were reported by Sannicandro,[56] and Rake[57] observed an endarteritis of the peripheral vessels in a case with vasomotor disturbances. Other observers have expressed the opinion that the primary lesion in scleroderma is in the walls of the blood vessels.[58-61]

Thus the basis for the conception that the sympathetic apparatus is of pathogenic significance in certain forms of scleroderma may be summarized briefly as follows: In scleroderma there frequently occur vasomotor disturbances which closely resemble Raynaud's phenomena. Environmental temperature has a pronounced influence on the vessels; exaggerated vasospasm follows exposure to cold and prompt vasodilation results from exposure to heat. Definitive vascular changes are observed histopathologically.

On the basis of these clinical and experimental observations which seemed to indicate a vasospastic element in scleroderma, Leriche[62] first proposed interruption of sympathetic impulses to the vessels as a therapeutic procedure. This was first performed by Brüning,[63] in 1923. Since then a number of observers have performed sympathectomy in scleroderma with varying results.[64-75] Some have reported remarkable improvements whereas others observed no beneficial changes. In an analysis of 13 patients in whom sympathectomy was performed, Leriche, Jung, and DeBakey[1, 2] observed marked improvement in five (38.4 per cent), moderate improvement in four (30.7 per cent), slight improvement in one (7.6 per cent), and no improvement in three (23 per cent). It was stated then that sympathectomy seems to be of value in the early cases in which the cutaneous lesions are not pronounced and in which the vasospastic phenomena are conspicuous.

ESOPHAGEAL DISTURBANCES

We have performed sympathectomy alone in one patient with scleroderma. This case was similarly studied according to the objective methods previously stated[3, 4, 5] and the results of these studies reveal marked improvement which has continued during the 17 months since operation. Of particular interest in this case are the esophageal manifestations. The patient developed progressive dysphagia during the last six months before the institution of surgical intervention. Fluoroscopic examination following the ingestion of a barium meal revealed retention at the cardiac end of the esophagus apparently due to a localized constriction. Esophagoscopy showed no growth but a stricture at the cardia and biopsy study of this tissue revealed normal mucosa. Since operation the dysphagia has

been considerably relieved. However, esophageal dilation has also been used.

Dysphagia and esophageal disturbances in scleroderma were first observed by Ehrmann,[76] in 1903. Since then, a number of cases have been recorded.[77-86] Weissenbach and his co-workers[82] state that including their four cases there have been a total of 18 cases reported in the literature, which clearly indicates its unusual occurrence. A more detailed study of this rare disturbance in scleroderma will be presented in a subsequent publication.[87]

SUMMARY

1. The surgical treatment of scleroderma consists of parathyroidectomy and sympathectomy.

2. The rationale of parathyroidectomy is based upon certain clinical and experimental observations which seem to indicate a definite physiologic disturbance in the calcium metabolism suggesting a hyperfunction of the parathyroids: (1) The presence of hypercalcemia in certain forms of scleroderma; (2) an abnormal increase of the calcium content of the skin in certain forms of scleroderma; (3) the occurrence of skeletal decalcification in some cases of scleroderma; (4) the experimental production of cutaneous changes clinically and histologically resembling scleroderma by the injections of parathyroid extract; and (5) evidence of hyperplasia and hypertrophy of the parathyroids in scleroderma.

3. The results of parathyroidectomy in previously reported cases are reviewed.

4. The rationale of sympathectomy is based upon certain clinical and experimental observations which seem to indicate that the sympathetic apparatus is of pathogenic significance in certain forms of scleroderma: (1) The frequent occurrence of vasomotor disturbances which closely resemble Raynaud's phenomena; (2) environmental temperature has a pronounced influence on the vessels; (3) exaggerated vasospasm follows exposure to cold and prompt vasodilatation results from exposure to heat; and (4) definitive vascular changes are observed histopathologically.

5. The results of sympathectomy in previously reported cases are reviewed.

6. We have performed parathyroidectomy and sympathectomy in three patients with scleroderma. The results of objective methods of study in these cases one and one-half years after operation reveal slight improvement in one, moderate improvement in the second, and marked improvement in the third.

7. The results in a fourth case in which sympathectomy alone was done reveal marked improvement seventeen months after operation.

8. The unusual complication of dysphagia and esophageal disturbances in scleroderma is described and occurred in one of our cases.

REFERENCES

1. Leriche, R., Jung, A. and DeBakey, M.: The surgical treatment of scleroderma, Surg., 1:6, 1937.

2. DeBakey, M.: Surgical treatment of scleroderma, New Orleans M. & S. J., 89:631, 1937.

3. Burch, G. E. and Sodeman, W. A.: A tonometer for measurement of tissue turgor in the human finger tip, Proc. Soc. Exp. Biol. & Med., 39:125, 1938.

4. Burch, G. E. and Sodeman, W. A.: The estimation of the subcutaneous tissue pressure by a direct method, J. Clin. Invest., 16:845, 1937.

5. Sodeman, W. A. and Burch, G. E.: A direct method for the estimation of skin distensibility with its application to the study of vascular states, J. Clin. Invest., 17:785, 1938.

6. Leriche, R. and Jung, A.: Vingt opérations parathyroïdiennes dans diverses affections, Rev. de Chir., 52:5, 1933.

7. Leriche, R.: A propos du traitement chirurgical de la sclérodermie, Bull. et mém. Soc. nat. de Chir., 58:7, 1932.

8. Leriche, R. and Jung, A.: Essai de traitement de la sclérodermie par la parathyroïdectomie, Bull. et mém. Soc. Nat. de Chir., 57:609, 1937.

9. Leriche, R. and Jung, A.: Nature et origine de la sclérodermie, Bulk Soc. franç. dermat. et syph., 42:885, 1935.

10. Leriche, R. and Jung, A.: Recherche sur la nature de la sclérodermie; les traductions tissulaires de l'hyperparathyroïdisme dans la sclérodermie; ostéolyse; surcharge calcique de la peau; signification des chiffres indiquant la teneur en calcium du sérum et des urines, Presse Méd., 43:1361, 1935.

11. Weissenbach, R. J., Basch, G., and Basch, M.: Les formes cliniques du syndrome de Thibierge-Weissenbach: concrétions calcaires des sclérodermies, Ann. dermat. et syph., 4:1, 1933.

12. Weissenbach, R. J., Basch, G., and Basch, M.: Les formes cliniques du syndrome de Thibierge-Weissenbach: concrétions calcaires des sclérodermies (IV. Etude Radiographique), Ann. de dermat. et syph., 4:125, 1933.

13. Weissenbach, R. J., Boppe; Martineau, J., and Malinsky, A.: Sclérodermie progressive. Télangiectasies multiples disséminées. Parathyroïdectomie, Bull. Soc. franç. de dermat. et syph., 42:1413, 1935.

14. Weissenbach, R. J., Boppe; Martineau, J., and Stewart: Sclérodermie progressive. Amélioration par la parathyroïdectomie, Bull. Soc. franç. de dermat. et syph., 43:1012, 1936.

15. Bernheim, A. R. and Garlock, J. H.: Parathyroidectomy for Raynaud's disease and scleroderma, Ann. Surg., 101:1012, 1935.

16. Garlock, J. H.: Parathyroidectomy for Raynaud's disease and scleroderma, S. Clin. North America, 16:771, 1936.

17. Durham, R. H.: A case of scleroderma with extensive subcutaneous, periarticular and vascular calcification, Ann. Clin. Med., 5:679, 1926.

18. Cornbleet, T. and Struck, H. C.: Calcium metabolism in scleroderma, Arch. Dermat. & Syph., 35:188, 1937.

19. Aisenberg, M. F.: Ueber Calcinosis interstitialis universalis, Fortschr. a. d. Geb. d. Röntgenstrahlen, 39:443, 1929.

20. Pernet: Sur un cas de sclerodermie avec calcinose, Arch. f. Dermat. u. Syph., 152:337, 1926.

21. Kennedy, R. L. J.: Calcinosis and scleroderma in a child treated by ketogenic diet, Med. Clin. N. Am., 12:1655, 1929.

22. Naegeli: A propos des sclérodermies, Bull. Soc. franç. de dermat. et syph., 36:883, 1929.

23. Pautrier, L. M. and Zorn: La calcémie dans les atrophies cutanées et dans les sclérodermies, Bull. Soc. franç. de dermat. et syph., 36:991, 1929.

24. Milian, G., Périn, L., and Horowitz: Sclérodermie calcaire, Bull. Soc. franç. de dermat. et syph., 37:475, 551, 1930.

25. Jung, A. and Hakki, A. C.: De l'influence des opérations parathyroïdienne sur la calcémie chez l'homme, Comp. rend. Soc. de biol., 108:40, 1937.

26. Weissenbach, R. J., Truchot, P., Françon, F., and Laudat, M.: Rheumatisme chronique fibreux, sclerodermie et concrétions calcaires sous-cutanées. Etude radiologique et bio-clinique, Bull. et mém. Soc. méd. d. hôp. de Paris, 47:837, 1931.

27. Jung, A. and Hakki, A. C.: Etudes sur la calcémie. Cent dosages du calcium sérique dans divers états pathologiques, affections osseuses et ostéoarticulaire, sclérodermie, retraction de l'aponévrose pulmaire, chéloïdes et autres, Rev. de chir., 51:537, 1932.

28. Michon, P. and Bohême, P.: Sclérodermie métamérique chez une hyperthyroïdienne. Hypocalcémie, Bull. Soc. franç. de dermat. et syph., 40:4, 1933.

29. Brooks, W. D. W.: Calcinosis, Quart. J. Med., 3:293, 1934.

30. Durham, R. H.: Scleroderma and calcinosis, Arch. Int. Med., 42:467, 1928.

31. Lereboullet, P. and Lelong, M.: Concrétions calcaires multiples de la peau avec sclérodermie localisée chez la mère et la fille, Bull. Soc. de pédiat. de Paris, 28:53, 1930.

32. Jaffe, H. L. and Bodansky, Aaron: Experimental fibrous osteodystrophy (ostitis fibrosa) in hyperparathyroid dogs, J. Exper. Med., 52:669, 1930.

33. Allbright, Aub, and Bauer: Quoted by Bernheim and Garlock, Ref. No. 15.

34. Selye, H.: A condition simulating human scleroderma in rats injected with parathyroid hormone, J. A. M. A., 99:108, 1932.

35. Shellings, D. H., Asher, D. E., and Jackson, D. A.: Calcium and phosphorus studies; effects in variations in dosage of parathormone and of calcium and phosphorus in diet on concentrations of calcium and inorganic phosphorus in serum and on histology and chemical composition of bones of rats, Bull. Johns Hopkins Hosp., 53:348, 1933.

36. Leriche, R., Jung, A., and Sureyya, C.: La peau dans l'hyperparathyroïdisme expérimental; étude de la sclérodermie experimentale, Presse méd., 43:777, 1935.

37. Hueper, W.: Metastatic calcifications in the organs of dogs after injections of parathyroid extract, Arch. Path. & Lab. Med., 3:14, 1927.

38. Brody, Joseph, and Bellin, D. E.: Calcinosis with scleroderma, Arch. Dermat. & Syph., 36:85, 1937.

39. Puddu, V.: Un caso di sclerodermia con calcificazioni operato di paratiroidectomia, Policlinico (sez. prat.), 41:1801, 1934.

40. Basch, G., Leibovici, R., Durupt, A., and Basch, M.: Sclérodermie avec concrétions calcaires (syndrome de Thibierge-Weissenbach) associée à une atrophie cutanée; parathyroidectomie, Bull. et mém. Soc. méd. d. hôp. de Paris, 50:516, 1934.

41. Spillmann, L., Hamant, A., Weis, J., and Créhange: Sclérodermie en plaques, troubles vasomoteurs et parathyroïdectomie, Bull. Soc. franç. de dermat. et syph., 42:991, 1935.

42. Brown, G. E. and O'Leary, P. A.: Skin capillaries in scleroderma, Arch. Int. Med., 36:73, 1925.

43. O'Leary, P. A. and Nomland, R.: A clinical study of one hundred and three cases of scleroderma, Am. J. Med. Sci., 180:95, 1930.

44. Lewis, T. and Pickering, G. W.: Observations upon maladies in which the blood supply to digits ceases intermittently or permanently, and upon bilateral gangrene of digits; observations relevant to so-called "Raynaud's disease", Clin. Science, 1:327, 1933-34.

45. Lewis, T. and Landis, E. M.: Further observations upon a variety of Raynaud's disease; with special reference to arteriolar defects and to scleroderma, Heart, 15:329, 1931.

46. Brown, G. E.: Skin capillaries in Raynaud's disease, Arch. Int. Med., 35:56, 1925.

47. Brown, G. E., O'Leary, P. A., and Adson, A. W.: Diagnostic and physiologic studies in certain forms of scleroderma, Ann. Int. Med., 4:531, 1930.

48. Pautrier, L. M. and Ullmo, A.: Note sur la capillaroscopie de la dermatite chronique atrophiante et de la sclérodermie, Bull. Soc. franç. de dermat. et syph., 36:776, 1929.

49. Thibierge, G. and Weissenbach, R. J.: Concrétions calcaires sous-cutanées et sclérodermie, Ann. de. dermat. et syph., 2:129, 1911.

50. Follmann, J. and Balló, B.: Beiträge zu den klinischen Untersuchungen und zur pathologischen Histologie der endokrinen Drüsen bei Sklerodermie, Dermat. Wchnschr., 97:1779, 1933.

51. Leriche, R. and Fontaine, R.: Le traitement chirurgical de la sclerodermie par les interventions sur le sympathique, Bull. Soc. franç. de dermat. et syph. 36:995, 1929.

52. Johnson, C. A. and Hedges, R. N.: Studies on peripheral vascular phenomena, Surg. Gynec. Obst., 60:1077, 1935.

53. Lewis, T. and Kerr, W.: Experiments relating to the peripheral mechanism involved in spasmodic arrest of circulation in the fingers, a variety of Raynaud's disease, Heart, 15:7, 1929-31.

54. Prinzmetal, Myron: Studies of the mechanism of circulatory insufficiency in Raynaud's disease in association with sclerodactylia, Arch. Int. Med., 58:309, 1936.

55. Matsui, S.: Anatomie pathologie et pathogenie de la sclerodermie generalisée, Mitt. a. d. med. Fak. d. k. Univ. zu Tokyo 31:53, 1924. Abst. Presse méd., 2:142, 1924.

56. Sannicandro, G.: Contributo alla conoscenza della sclerodermia con speciale riguardo ai suoi rapporti con le turbe metaboliche del calcio, Arch. ital. di dermat., sif. 4:427, 1929.

57. Rake, G.: On the pathology and pathogenesis of scleroderma, Bull. Johns Hopkins Hosp., 48:212, 1931.

58. Dinkler, M.: Zur Lehre von Sclerodermie, Deutsches Arch. f. Klin. Med., 48:514, 1891.

59. Newman, M. F.: Handbuch der Haulkrankheiten, A. Hölder, Vienna, 1904.

60. Crocker, H. R.: Diseases of the Skin. Their Description, Pathology, Diagnosis, and Treatment, P. Blakiston's Son and Co., Philadelphia, 1908.

61. Gaucher, E.: Maladies de la peau, J. B. Baillière et fils, Paris, 1909.

62. Leriche, R.: Sur l'étude experimentale, la technique et quelques indications nouvelles de la sympathectomie périartérielle, Presse méd., 30:1105, 1922.

63. Brüning, F.: Ueber Dauererfolge und Misserfolge der periarteriellen Sympathektomie, insbesondere über ihre Ausführung bei der arteriosklerotischen Gangrän, Klin. Wchsnhcr., 2:923, 1923.

64. Enderlen: Zentralbl. f. Chir., p. 1833, 1922.

65. Horn: Ueber periarterielle sympathektomie bei Sklerodermie, Zentralbl. f. Chir., 50:831, 1933.

66. Kappis: Weitere Erfahrungen mit der Sympathektomie, Klin. Wchnschr., 2:1441, 1923.

67. Stahl: Spatergebnisse der periarteriellen Sympathektomie mit einigen Bemerkungen über die Ernahrung der Arterienwande, Deutsche Ztschr. f. Chir., 197:280, 1926.

68. Kment, H.: Ergebnisse der Brüning-Leriche'schen Operation, Beitr. z. klin. Chir., 140:220, 1927.

69. Lehmann, W.: Die Dauererfolge der periarteriellen Sympathektomie, Beitr. z. klin. Chir., 143:320, 1928.

70. Muller, G. P.: End results of periarterial sympathectomy, Ann. Surg., 88:474, 1928.

71. Adson, Alfred W., O'Leary, Paul A., and Brown, George E.: Surgical treatment of vasospastic types of scleroderma by resection of sympathetic ganglia and trunks, Ann. Int. Med., 4:555, 1930.

72. Leriche, R., Wolf, M., and Fontaine, R.: Résultats d'interventions sur le sympathique dans la sclérodermie, Bull. Soc. franç. de dermat. et syph., 32:153, 1925.

73. Leriche, R. and Fontaine, R.: Résultats un peu éloignés des interventions sur le sympathique dans la sclérodermie et dans la dermatite chronique atrophiante, Rev. de chir., Paris, 65:285, 1927.

74. Leriche, R. and Robineau: Indications et résultats de la sympathectomie périartérielle dans la chirurgie des membres, Paris chir., 19:191, 1927.

75. Gaté, J., Michel, P. J., and Cuilleret, P.: Sclérodactylie progressive avec syndrome de Raynaud, opérée en 1933 et 1934. Résultats thérapeutiques, Bull. Soc. franç. de dermat. et de syph., 42:1032, 1935.

76. Ehrmann: Ueber die Beziehung der Sklerodermie zu den autotoscischen Erythemen, Wiener med. Wchnschr., 6:6, 1903.

77. Schmidt, R.: Sklerodermit mit Dysphagie, Med. klin., 12:460, 1916.

78. Schwarz, P.: Sklerodermie und Röntgenkastration, Schweiz. med. Wchnschr., 56:246, 1926.

79. Nomland, R.: Sclerodactylia with calcification, Arch. Dermat. & Syph., 21:322, 1930.

80. Fessler, A. and Pohl, R.: Stenosierender Progess des Oesophagus bei Sklerodermie, Dermat. Ztschr., 63:164, 1932.

81. Kuré, K., Yamagata, K., Tsukada, S., and Hiyoshi, J.: Passagestörung des Oesophagus bei Sklerodermie und Dystrophia musculorum progressiva, Klin. Wchnschr., 15:516, 1936.

82. Weissenbach, R. J., Stewart, W., and Hoesli, H.: Les troublés fonctionnels oesophagiens et les lésions de l'oesophage dans la sclérodermа, Bull. Soc. franç. de dermat. et syph., 44:1060, 1937.

83. Cailliau: Discussion of Weissenbach, Stewart, and Hoesli, Bull. Soc. franç. de dermat. et syph., 44:1060, 1937.

84. Weissenbach, R. J., Martineau, J., Bouwens; Pizon; and di Matteo: Sclérodermie progressive. Sydrome de Thibierge-Weissenbach. Ulcère de jambe et calcification en molletières. Troubles oesophagiens, Bull. Soc. franç. de dermat. et syph., 44:2018, 1937.

85. Hoesli, Henri: Troubles fonctionnels et lésions de l'oesophage dans la sclerodermie, Thèse de Paris, 1937, Jouve et Cie, Paris.

86. Weissenbach, R. J., Dechaume; Martineau, J., Stewart, W., and Hoesli, H.: Un case de syndrome de Thibierge-Weissenbach (sclérodermie progressive et calcifications) avec lésions bucco-pharyngéis et oesophagiennes accentuées, ayant nécessité la gastrostomie, Bull. Soc. franç. de dermat. et syph., 44:1053, 1937.

87. Ochsner, Alton and DeBakey, Michael: Esophageal manifestations of scleroderma. Review of literature and report of an additional case. In press.

A PRELIMINARY REPORT ON A PRESUMPTIVE SIGN OF SYPHILIS

ALFRED L. ADAM, M. D.†
SHREVEPORT, LA.

There are many signs and symptoms by which syphilis may be detected, the most notable of which are the darkfield, the Wassermann test and the x-ray findings. Among the lesser signs are those produced by characteristic skin, bone and nerve lesions of early and late syphilis familiar to every one. The enlargement of the epitrochlear lymphatic glands is a fairly constant finding and is in a way related to the sign to be presented.

The clinics in Caddo Parish have a case load of about 1,200 patients and ample opportunity is presented, particularly in the Shreveport clinic, to study and evaluate signs and symptoms of early and late syphilis.

THE SPECIAL SIGN

A consistent sign that I have noted here is an erythematous inflammatory condition of the buccal orifice and intramural portion of Stensen's duct. The intramural part of the duct, that is, the terminal part of the duct that lies between the buccinator muscle and the mucous membrane is not considered as important as the appearance of the orifice proper. The changes in the appearance of the duct orifice need careful interpretation to be of maximum value if my deductions are correct. Furthermore, it might be well to remember that varying climatic conditions with varying upper respiratory tract infections, the variations in oral hygiene, variations in diet and constitutional diseases may well change the value of this sign. In this locality the dental conditions are excellent, the diet fairly well balanced and throat, mouth and ear infections are few and far between.

A brief description of the sign as I have observed it and a possible explanation of the evident pathology follows. In almost all early cases of syphilis and many of late

†From the Caddo-Shreveport Health Unit, Venereal Disease Division.

syphilis I have noted the following changes in Stensen's duct:

1. Edema and congestion or swelling of the duct and adjacent area.

2. A flap-like fold of mucous membrane about the duct accentuated by the edema or congestion.

3. An inflammation of the orifice.

1. The edematous congestion of the duct and adjacent area must be interpreted very carefully and is probably not of very great significance when occurring alone. Bad oral hygiene; carious teeth, particularly upper second molars; use of snuff; leukoplakial patches, and chronic pharyngitis tend to cause this condition, I believe. The use of snuff placed in the cheek and held there like chewing tobacco is a common habit here among the negroes.

2. The flap-like fold of mucous membrane is considered a normal departure from the usual anatomy and has about the same slight significance as the above simple congestion of the duct in the adjacent area.

3. The inflammation about the orifice with or without congestion is considered the major phase of the sign.

The following subdivisions of the sign have been noted: (a) A pinhead sized lesion about 2 mm., well circumscribed and of pale pink to reddish cast; (b) a red, erythematous inflammatory lesion slightly larger than the above, of greater degree of redness and not well demarcated; (c) a very red lesion, with slight suggestion of purplish color, diffuse and from two to five mm. in size, usually with congestion.

The pinhead lesion and the not well demarcated lesion are seen often enough in negative cases so as to be considered only slightly presumptive but when evaluated with history and physical findings it strengthens somewhat the suspicion of syphilis.

The lesion mentioned under heading (c) is the crux of the entire sign but like meatoscopy of the ureteral orifice, ophthalmoscopy and examination of the cervix, it needs careful examination and interpretation. With the diffuse, very red condition of one or both orifices, without a chancre and negative Wassermann, it might be well to keep the patient under observation especially if the epitrochlears can be palpated.

An attempt in the following paragraph is made to show why such a sign should be present and why it should be of more value than epitrochlear enlargement. Let us first consider the anatomy of the parotid gland and remember the anatomy and pathologic changes of the ureter, and the anatomy and pathology of the prostate with its ejaculatory ducts. In the latter two organs certain types of pathology manifest themselves in changes in the ureteral orifices and the openings of the ejaculatory ducts in the posterior urethra. It might be better to compare what we know of kidney pathology with its obstructive uropathic changes to the conditions present in the parotid gland and duct in syphilis. In the kidney, obstruction with stasis of urine resulting from calyceal or pelvic deformity caused by pyelonephritis, cyst, stones, and tumors or aberrant blood vessels and ptosed kidneys frequently gives rise to infection, either a new type or superimposed on inflammatory conditions already present.

In the case of the parotid gland, the presence of lymphatic glands in the substance of the parotid is presumed to take part in the general adenopathy of syphilis and through persistence of their enlargement, such as in the cases of the epitrochlears, cause in course of time some macroscopic evidence of inflammation about the orifice of Stensen's duct because of mechanical interference with the flow of salivary fluid from the gland. Ascending, lymphatic or hematogenous infections of low grade character apparently result as evidenced by the sign being described in this paper. Some bacteriostatic phase of the salivary secretion may hold this infection in abeyance or limit it to such a low degree that it escapes notice.

To support the above thought a brief quotation from Morris' Anatomy is given: "Finally, there are embedded in the gland two or three deep lymphatic nodes, which

receive lymphatic vessels from the external meatus, the soft palate, and the posterior part of the nasal fossa, and several superficial nodes which receive lymphatic vessels from the temple, eyebrows and eyelids, cheek and auricle."

Retrograde x-ray of the parotid duct and its branches could be made and studied using skiodan or uroselectan as in pyelography and possibly some light may be brought to bear upon this subject.

Interpretative and technical difficulty should be anticipated due to the fine caliber of the beginning duct. Cultures, rates of flow and changes in the composition of the salivary secretion with regard to relative amounts of water, salts and the organic material obtained through stimulation of the chorda tympani nerve in these cases may well give different results from the normal, provided the lympathic glands enlarge sufficiently to affect the secretory mechanisms.

SUMMARY

1. A sign persistently present in our cases has been described.
2. Anatomic and pathologic explanation has been attempted.
3. Other tests that might re-enforce the sign have been suggested.

CONCLUSIONS

If other investigators coincide with my findings and the sign does exist in a fair degree of patients in all localities, I believe due to its accessibility it should prove of definite value and certainly no harm can result from suspected syphilis in every patient. If this paper only stimulates interest in carefully studying patients for infectious syphilis, early cardiovascular, and early neurosyphilis, it will be of definite value.

DIAGNOSIS AND TREATMENT OF BRONCHIAL ASTHMA*

NICHOLAS K. EDRINGTON, M. D.
NEW ORLEANS

The diagnosis of bronchial asthma is based upon many points that are characteristic of the asthmatic in a spasm. If the patient is seen during the attack the diagnosis is comparatively simple. The characteristic orthopneic position and the wheezing expiratory type of respiration are in most cases sufficient evidence upon which to make this diagnosis. However, in the interval or free stage the diagnosis is sometimes rather difficult, due to the fact that the examination reveals no characteristic abnormalities and it is here that one has to depend upon the patient's history. Thus if a patient gives a history of paroxysmal, periodic or recurrent attacks of acute dyspnea, accompanied by wheezing and especially occurring at night, it is safe to make a diagnosis of asthma. A history of a preceding or associated hay fever or coryza or a sensitivity to some food and a family history of allergy strengthen greatly the presumptive diagnosis of asthma.

In a small percentage of patients the history may be so indefinite that the diagnosis is sometimes difficult. Occasionally some complicating condition may so mask the underlying or basic disease as to make it hard to determine its real nature, or factors residing outside of the bronchial tree may produce symptoms simulating asthma. In these instances the diagnosis must of necessity be differential in character and can be greatly aided by the finding on examination of the patient of the responsible organic lesion or by the use of laboratory methods or other special examinations.

Faced with a problem of doubtful allergic etiology, certain diagnostic criteria may be applied which were propounded by Rackemann:[1]

"1. A presenting symptom which can be explained by smooth muscle spasm, or by increased capillary permeability.

"2. The occurrence of one or several other manifestations of allergy.

*Read at Baptist Hospital Clinic of the Annual Meeting of the American College of Physicians, New Orleans, March 30, 1939.

"3. A positive family history of allergy.
"4. The presence of positive skin tests.
"5. The presence of a blood eosinophilia."

The indication that allergy must be considered depends upon the greater number of the above characteristics present in the patient's symptoms. To these criteria may be added the clinical response to epinephrine, which furnishes an important therapeutic guide in many cases.

When it has been established that we are dealing with an allergic problem, it is now necessary to establish the importance of certain specific causative or exciting factors and the secondary or predisposing factors that may have a bearing on the case. For this purpose, we must depend not only upon the history, physical examination and laboratory tests, but also upon specific skin and other tests for allergens, therapeutic trial and certain special examinations when indicated.

IMPORTANCE OF THE HISTORY

A detailed and carefully taken history constitutes one of the most important aids in the diagnosis of bronchial asthma. In fact, in many instances, diagnosis can be made on the history alone. While the patient is being questioned the necessity for determining the many factors must be constantly borne in mind. Information must be elicited as to the mode and date of the initial attack, which will often bring out valuable clues. Should the description of the attacks be similar, the history of a typical one is sufficient, but frequently the sequence of events is helpful. The time when attacks occur, whether seasonal or perennial, is very important.

The environment of the patient and the time of day when symptoms first appear should be carefully noted. The duration of the attacks, their frequency and severity and the effect of change of environment and medication will give further information. The amount of relief between attacks is of considerable prognostic value. Careful inquiry should be made to bring out the part that some primary or aggravating factor may play in the case. Thus, an attack of asthma may follow an infection, such as a cold, or exposure to house dust, animal pets, the use of cosmetics, or the ingestion of certain foods. Occupation and environment of the patient sometimes are responsible for the exciting factors; such as the baker with wheat, the farmer with cattle, horses, chickens and grain dust, the beautician with orris root and cosmetics. Climatic conditions such as moisture, dryness, wind or changes in barometric pressure may be important and physical agents such as heat and cold may have some relation to the attack. The length of time the patient has resided in his present quarters and the effect of any recent change should be brought out. Careful questioning should elicit a description of the house furnishings, especially those of the bedroom, for the presence of possible allergens may be found in the rugs, carpets, bedding, upholstery of the furniture and contents of clothes closets. The presence of pets, such as a cat or dog about the home and the kind of soaps, tooth powders and cosmetics being used should be disclosed.

The patient should be questioned as to what effect a vacation or a change of habits or environment has on symptoms. Prolonged hospitalization often results in the clearing up of the symptoms of bronchial asthma due, no doubt, to the removal of the patient from an environment containing the exciting allergen.

A history of manifestations of allergy in the past, especially in childhood, should be carefully sought. Recurrent colds or cases of bronchitis, urticaria, migraine, cyclic vomiting or eczema greatly assist in establishing an allergic background.

A careful and systematic review of the general health of the patient should be made. The existence of anemia, endocrine disturbances, increased nervous tension, digestive upsets, foci of infection, menstrual disorders and inadequate rest are factors of importance, the consideration and treatment of which are essential to achieve the best results from therapy. Any so-called idiosyncrasy to drugs such as phenolphthalein, aspirin, or quinine, should be looked

upon with suspicion, for allergy to certain drugs is probably much more common than is generally supposed.

One of the outstanding facts concerning the allergic patient is that he possesses a constitution different from his fellowmen. That this allergic constitution is inherited from one generation to another is sufficiently established by the studies of Clark, Donelly, Coca,[2] Balyeat[3] and others. Studies indicate that what is inherited is not the specific disease itself, but the allergic tendency which makes the descendants subject to become sensitive to anything. A positive family history of allergy forms a very useful criterion in doubtful cases. Investigation of the "family tree" should include at least four generations: the grandparents, the parents, uncles and aunts, brothers, sisters and cousins. A history of "catarrh," sinusitis, bronchitis, hives and eczema in other members of the family, though masked by the patient by other names, should be considered with suspicion.

Asthmatic patients, apart from their manifestations of allergy, are usually healthy individuals; nevertheless, one should never omit the physical examination. A careful check up should be made of the eyes, nose, mouth, chest and skin. Attention should be directed to the possibility of focal infection in the sinuses, teeth, tonsils, prostate and bronchial tubes. Often it is necessary to differentiate cardiac from bronchial asthma; therefore, the heart should not be neglected in the examination. It is essential to note the general state of nutrition of the patient, nervous state and evidence of any metabolic disturbance.

LABORATORY EXAMINATIONS

Laboratory examination of the patient with bronchial asthma should include the routine examination of the urine, a complete blood count with special emphasis placed on the differential study of the white blood cells for eosinophils. The presence of a large number of eosinophils in the blood smear and in the bronchial secretions is additional evidence that the condition being investigated is of allergic origin. Continued absence of eosinophils, coupled with the presence of a large number of neutrophils, suggests an infection rather than an allergic process. In 1934 Vaughan[4, 5] revived Widal's "digestive hemoclasia" test, renamed it the "leukopenic index," and has since been advocating it as a diagnostic aid in food allergy. However, the more recent work of Loveless, Dorfman and Downing[6] and others has demonstrated that a postprandial leukopenia was not found to be an index to incompatible foods. In cases where an element of infection is suspected, the erythrocyte sedimentation rate is of some value. The metabolic rate, gastric analysis and additional studies of the blood chemistry are not infrequently indicated. X-ray examination of the chest following the intratracheal instillation of iodized oil should be made to exclude the presence of bronchiectasis. Examination of the sputum for the tubercle bacilli is, of course, made in doubtful cases and an autogenous vaccine may be made from the sputum when the symptoms seem largely explainable on a basis of infection.

SKIN TESTING

While the diagnosis of allergy in most cases is made on the basis of the history and is confirmed by physical and laboratory findings, it is essential that skin tests be performed mainly in an effort to determine the specific exciting agents; however, reliance on them alone is apt to be misleading. One must remember a positive skin test does not always indicate that the patient is clinically sensitive to the allergen in question. A positive skin test will reveal, not only present sensitivities, but sensitivities existing and persisting from childhood, or may represent future sensitivity. Negative skin tests do not mean that the patient is not allergic, and finally, the size of the test reaction is not necessarily correlated with the degree of sensitivity. It must be emphasized then, that, if skin tests are to be made, they should be adequate thoroughly to investigate the particular case, bearing in mind that the reactivity of the skin varies markedly in different patients. Finally, the results obtained from the skin test

should be carefully evaluated in the light of the clinical history.

Where foods may be suspected as a factor, and skin tests are negative, or for some reason cannot be performed, the use of elimination diets should be employed. If, following the elimination of some food, the symptoms disappear only to return when the specific food is placed back in the diet, it is possible to incriminate this food as a probable factor.

TREATMENT

The most effective therapeutic measure for the relief of the asthmatic paroxysm is the administration of epinephrine (1:1000 dilution) by hypodermic injection. Epinephrine produces its effect by heightening the tone of the sympathetic component of the vegetative nervous system. In the lungs it overcomes the opposing parasympathetic or vagus pull, the bronchospasm is relieved and the edema and swelling of the bronchial mucosa are reduced to vasoconstriction of the arterioles. There are practically no contraindications to the use of epinephrine. It is not injurious to the body tissues, nor is it a habit-forming drug. The dose necessary to reduce the spasm depends upon the age of the patient, the severity of the attack and whether or not the patient has had previous injections of the drug. Usually from two or five minims of the drug suffice to bring relief within a few minutes, and the earlier in the attack the drug is given, the smaller the dose required. A most common error is to give an initial dose of 1 c.c. since this represents the contents of the usual ampule, with the result that many patients suffer from alarming and annoying symptoms such as extreme pallor, marked tremor, palpitation, throbbing headache, nervousness and weakness which may persist for quite a while. Such symptoms do not endanger the life of the patient; however, the discomfort often discourages subsequent administration of the drug.

In the more chronic cases, especially those of many years' duration, the dose of epinephrine may have to be repeated and larger doses (2-3 c.c.) may have to be given before the attack is broken. Once the severe attack has subsided, it will be found that the patient can again be relieved by the smaller or usual dose. When the asthmatic attacks occur at frequent intervals, it is advisable to instruct the patient or some member of the family as to the technic of epinephrine injection in order to permit its administration as often as required and as soon as possible in the onset of the attack. This procedure may mean the difference between a good or bad night for the patient. The use of epinephrine in 1:100 dilution by oral inhalation has been used for quite a few years. My enthusiasm for this method for the relief of the asthmatic attack soon waned, because I found, in many instances, the desired relief was not obtained due to the insufficient amount of vapor produced by the nebulizers available for this purpose. More recently the DeVilbiss Company has put on the market an all-glass nebulizer No. 40 for vaporizing concentrated epinephrine and other aqueous solutions. This apparatus produces approximately five times the amount of vapor as the older types and can be adjusted to regulate the vapor to a lesser amount. Results in the control of the asthmatic paroxysm with this type of nebulizer has produced excellent results and has greatly reduced the number of injections of epinephrine. Contrary to expectation, instances of over dosage with the more powerful vaporizers are very seldom encountered. Dryness of the mouth and pharynx can be avoided by having the patient rinse the mouth with water following the use of the vaporizer. Ephedrine is a fairly efficient substitute for epinephrine and has the advantage that it can be administered orally. Its action, however, is not so prompt and efficient as epinephrine and in many patients produces somewhat disagreeable after effects, such as nervousness and insomnia. The ephedrine preparation most commonly used is the sulphate or hydrochloride in combination with amytal or phenobarbital, the latter drugs to counteract the after effects of the ephedrine. Propadrine hydrochloride, which possesses none of the after effects

of ephedrine, used in combination with theophylline, makes an excellent preparation for the control of the attacks.* Morphine should should never be used in patients in whom epinephrine effectively relieves the attack. It is a dangerous drug in asthma and probably should never be used. Clinically, asthmatic patients of any age, especially children and elderly people whose bronchial tubes are filled with mucus, should not have morphine given for the relief of their symptoms. Morphine paralyzes the cough reflex and depresses the respiratory center, defeating nature's efforts in relieving the bronchial tubes of the tenacious mucus.

In the more severe cases of intractable asthma, or the cases of so-called status asthmaticus, where repeated doses of epinephrine have failed to control the paroxysm, the use of aminophylline U. S. P., or theophylline ethylenediamine in 7½ grain doses, mixed with 10-20 c.c. of glucose solution given slowly and intravenously seems to be most effective, prompt, reliable, safe and, at times, apparently life-saving therapeutic measure. Should this fail, rectal ether narcosis should be tried. Equal parts of ether in olive oil (5-7 ounces for adults and ½ to 2 ounces for children) should be given slowly through a rectal tube. Helium 80 per cent and oxygen 20 per cent applied by inhalation with a special closed apparatus has produced definite improvement.

The measures outlined provide in most patients immediate and complete relief of the acute asthmatic symptoms. In the long standing or chronic cases, especially those with a superimposed bronchitis and those with more or less continuous asthma, symptomatic treatment is likewise necessary in addition to that employed during the acute attack. A combination of expectorants, antispasmodic and sedative drugs are employed for this purpose. Potassium or sodium iodide in 5-10 grain doses, lobelia and stramonium, 5-10 minim doses of the tincture and the barbiturates in ¼ - ½ grain doses are valuable drugs.

Autogenous bacterial vaccines prepared from the nasal and bronchial secretions should be used in all cases where a secondary bronchial infection exists. The question as to whether their administration constitutes a form of specific or non-specific desensitization is academic and controversial. However, clinical experience has proved these vaccines of beneficial value. Attempts should be made to improve the general health of the patient. Foci of infection should be removed or corrected, if they are either detrimental to the general health of the patient, or of importance in relation to the asthmatic condition. Treatment of abnormalities of the nasal passages and of infections of the paranasal sinuses should be conservative; radical measures should be avoided unless all other methods to bring relief have failed.

The correction and restoration to a normal state of body chemistry and endocrine balance is quite important in the management of the asthmatic person. Hygienic measures which will serve to improve the well-being of the patient should be applied to the chronic asthmatic, especially during the free or interval stage. Heavy meals in the late evening should be avoided as gastric distention may provoke an attack. The principal meal should be taken at noon. Bowels should be properly regulated to avoid constipation and flatulence. Indigestion and dyspepsia sometimes may be the result of a deficiency of hydrochloric acid in the gastric juice. In these cases, dilute hydrochloric acid in 15-60 minim doses should be administered before meals.

The specific treatment of asthma, as with other allergic conditions, is dependent upon the etiologic factors brought out by diagnostic study. It consists of the elimination of, or the avoidance of contact with the specific excitants. Pillows and mattress may be covered with an allergen proof encasing; dust in the bedroom may be prevented by making the bedroom as barren as possible and by using washable rag rugs. Pets

*Tablets containing propadrine hydrochloride, gr. ½, and theophylline, gr. 2, were supplied for experimental use through the courtesy of Sharp & Dohme. These tablets are now on the market under the name of padrophyl.

should not be allowed in the house and non-allergic cosmetics should be used. All food giving definite positive reactions and found on clinical trial to be a factor in the case should be removed from the diet.

Desensitization therapy by subcutaneous injections with extracts of the inhalant allergens should be instituted when the reactions to them are markedly positive and when contact cannot be avoided easily, as in the case of house dust, molds, orris root, or the animal danders, and when the pollens give positive reactions.

Patients suffering from periodic attacks of severe asthma, with mild daily attacks between with the production of considerable white glary or semi-purulent mucus are often greatly benefited by the intratracheal instillation of iodized oil. Its use cannot in itself cure a patient suffering from allergic asthma, but is of inestimable value as a means of dislodging and forcing up bronchial plugs and tenacious mucus and replacing pockets of pus with a non-irritating substance.

SUMMARY

It is extremely difficult to estimate definitely the results of treatment in asthma. All that can be said, in a general way, is that the results obtained by the present day treatment are a vast improvement over those of 20 to 30 years ago, and the outlook for the asthmatic patient is not so hopeless now as it was in former years. Time and a better understanding of many factors concerned will accomplish much towards improving the results of treatment.

REFERENCES

1. Rackeman, F. M.: Clinical Allergy, Asthma and Hay Fever. New York: The Macmillan Company, 1931, p. 225.

2. Clarke, J. A. Jr., Donnally, H. H., and Coca, A. F.: Studies in specific hypersensitiveness; on influence of heredity in atopy, J. Immunol., 15: 9, 1928.

3. Balyeat, Ray M.: The hereditary factor in allergic diseases, Am. J. Med. Sci., 176: 332, 1928.

4. Vaughan, W. T.: Further studies of the leukopenic index in food allergy, J. Allergy, 6: 78, 1934.

5. ———— The leukopenic index as a diagnostic method in the study of food allergy, J. Lab. & Clin. Med., 21: 1278, 1936.

6. Loveless, Mary; Dorfman, Robert; and Downing, Lillian: A statistical evaluation of the leukopenic index in allergy, J. Allergy, 9: 321, 1938.

TORSION OF THE GALLBLADDER

REPORT OF A CASE, WITH A BRIEF COMMENT ON CERTAIN ASPECTS OF THIS ACCIDENT*

H. REICHARD KAHLE, M. D.
NEW ORLEANS

CASE REPORT

A. V., 60 years of age, a white farmer of French descent, was admitted to Charity Hospital of Louisiana at New Orleans shortly after midnight March 18, 1938. The history was unsatisfactory because the patient was in too much pain, and, as a result, too restless to be cooperative.

His illness had begun several days before admission with mild pain in the mid epigastrium. The pain radiated into the right lower quadrant, and was not relieved by two bowel movements which followed the taking of epsom salts shortly after the onset. Intermittent nausea had been present through the illness, but the patient had not vomited at any time. Seven hours before admission the pain became very acute. The physician who was then consulted for the first time made a diagnosis of acute appendicitis and advised immediate hospitalization. At the time of admission the pain was very severe and was localized in the right lower quadrant. There were no other symptoms except slight difficulty in voiding. No relevant facts were elicited from the previous history.

The temperature on admission was 98° F., the pulse 60, the respiratory rate 16, and the blood pressure 140/110. Except for a few sibilant and sonorous rales throughout the chest, examination of the head, neck and chest was entirely negative. The respiratory movements of the abdomen were limited. There was marked rigidity in the epigastrium and in the right lower quadrant, which could be somewhat relaxed after a little effort. No organs or masses could be palpated, and no herniae were present.

Urinalysis revealed nothing abnormal. The white blood cells numbered 16,500, with 72 per cent neutrophils.

Although the normal pulse rate and temperature were against such a diagnosis, the most likely possibility seemed to be acute appendicitis. Ruptured peptic ulcer, with spillage along the right lumbar gutter, was also considered, although no history of previous dyspepsia could be obtained.

As the condition was clearly surgical, the abdomen was opened under nitrous oxide and ether anesthesia an hour and a half after the patient was admitted. A right transrectus incision was

*From the Department of Surgery of the School of Medicine of Louisiana State University, and Charity Hospital of Louisiana at New Orleans.

used, with its center opposite the umbilicus. No fluid was found in the peritoneal cavity, nor was there any evidence of peritonitis. The appendix was entirely normal.

Further exploration revealed a brownish-red mass, which, when the incision was extended upward, proved to be the gallbladder. It was very much distended and buckled at the liver edge. This finding led to the suspicion that torsion had occurred. Further examination showed that the gallbladder, which was suspended from the under surface of the liver in an avascular peritoneal fold resembling a mesentery, had completely twisted on this attachment in a clock-wise direction. No point of perforation could be detected, although there was a slight brownish stain on the anterior wall of the stomach, where the mass had been in contact with this organ.

The torsion was reduced and cholecystectomy done without difficulty, since the peritoneal attachment was avascular and the gallbladder mobile. The pedicle containing the cystic duct and vessels was divided and doubly ligated. The appendix was not disturbed. A drain was left in the region of the cystic duct and brought out through a stab wound. The abdomen was closed in layers.

The gallbladder was desired as a museum specimen and was therefore not opened, but no stones could be palpated within it.

Convalescence was without incident except for a psychosis which began on the fifth day and persisted until the ninth. The patient was discharged in excellent condition on the twelfth day, and according to his family physician has remained well to date, thirteen months after operation.

COMMENT

The first case of torsion of the gallbladder was reported by Wendel in 1898. Some 70 cases are now on record, including the two just reported by Berry[3] in 1939. Most of them are in the foreign literature, and two or three others may be reported in certain of these articles not available at this time. Shipley,[7] in 1927, and Short and Paul,[6] in 1934, have so thoroughly covered the subject that another detailed study would not be justified, though the condition is still sufficiently infrequent to warrant the report of single cases as they occur.

Except for the fact that in the case herewith reported the patient was a man, it presented no unusual features. In the 60 cases in which information as to sex of the patient is available, only eight were men.

Typically the patient is a narrow-waisted, elderly female who is suddenly stricken with colicky pain in the midabdomen or right hypochondrium. Vomiting is frequent but jaundice is rare. The temperature and pulse remain normal for a long period of time, in spite of the extremely severe pain. Symptoms of peritonitis supervene on the original symptoms only when the gallbladder wall becomes gangrenous and perforates. Until that time, the most striking characteristics of the illness are the intense pain and the presence of a mass, which may disappear and reappear, in the upper right quadrant, the alterations probably being due to the twisting and untwisting of the pedicle.

The accident is usually considered to be caused by abnormal mobility of the gallbladder, either as the result of an absence of attachment or a loose attachment to the under surface of the liver, or the presence of a mesentery in which the gallbladder is suspended. Peristaltic movements in the transverse colon have been considered as a possible exciting cause. In the case reported in this paper, the attack of pain began before the patient took a purgative, but was much worse afterwards, and perhaps the increased intestinal movements did play some part. If, however, purgation had been the chief etiologic factor, in all probability the rotation would have been counter-clockwise instead of clockwise. A counter-clockwise movement seems to be rather more common in the reported cases.

Stones are frequently found in gallbladders which have undergone torsion, but are thought to have little to do with causing the accident. In Short and Paul's opinion they are present no more frequently in such cases than one would expect to find them in elderly women in general.

The mortality is surprisingly low (16 per cent, according to Arthur[2]) considering that the patients are usually advanced in years. The ease with which cholecystectomy can be performed, because of the loose attachment of the gallbladder to the liver, probably explains most of the favorable results.

Torsion of the gallbladder is most often diagnosed as acute cholecystitis or cholelithiasis, followed in the order of frequency by appendicitis and intestinal obstruction.

A number of authors consider the clinical picture distinct enough to permit a correct preoperative diagnosis, which has been made in a number of the reported cases. It seems fair enough to say that if the accident is borne in mind, a dramatic diagnosis will often be possible. As Berry points out, however, the condition is so very unusual that the likelihood of its presence is rarely considered.

SUMMARY

A case of torsion of the gallbladder is added to the approximately 70 cases already on record. A brief comment is made on several important features of the accident, and it is pointed out that a preoperative diagnosis could frequently be made if the possibility of its occurrence were borne in mind. The mortality is surprisingly low considering the advanced age of most of the patients.

REFERENCES

1. Angel, F., and Angel, E.: Torsion of the gallbladder with gangrene; case report, South. M. J., 29:944, 1936.

2. Arthur, H. R.: Acute torsion of gallbladder, Brit. M. J., 2:265, 1937.

3. Berry, W. H.: Torsion of the gallbladder, J. A. M. A., 112:1580, 1939.

4. Blank, E.: Torsion of gallbladder with perforation and gangrene in boy 17, New England J. Med., 212:197, 1935.

5. Hun, H. H.: Torsion of the gallbladder, Albany M. Ann., 55:98, 1936.

6. Short, A. R., and Paul, R. G.: Torsion of the gallbladder, Brit. J. Surg., 22:301, 1934.

7. Shipley, A. M.: Torsion of the gallbladder, Arch. Surg., 14:968, 1927.

8. Suggit, S. C.: Torsion of the gallbladder, Brit. M. J., 1:977, 1935.

PATHOLOGIC FRACTURE OF RIB

REPORT OF A CASE

A. MAYORAL, M. D.
NEW ORLEANS

Pathologic fractures are well known, but they are by no means common. Within the past four years I have seen only one, and this of a very unusual nature.

This type of fracture, sometimes called spontaneous, always follows slight external violence, or muscular action of less degree. Clinically they are found to be due to inflammatory bone processes; neoplasms, benign or malignant; bone atrophy of disuse, or imperfect osteogenesis.

Syphilis, mentioned by some as a cause of fractures, is extremely rare except in cases of tabes.

The case presented is one of gumma of the left eighth rib.

CASE REPORT

G. T., a 51-year-old Norwegian, white seaman, was admitted to the Marine Hospital on September 26, 1932. The diagnosis was syphilis, tertiary, general. The family history was unimportant. He denied previous diseases except a penile lesion in 1907. No treatment had been received until October, 1930, when patient received five intravenous injections of neoarsphenamine in San Pedro, California. On July 15, 1932, while in Calcutta, India, he noticed a pain in the lower chest. Since then he has been having intermittent shooting pains in the chest, at times severe. A shipmate applied a mustard plaster to his chest, which caused a severe burn. This burn brought him to the hospital.

Skiagram of the chest revealed pathologic fracture (fig. 1) of the eighth left rib. Although his syphilitic condition was known, extensive work was done to rule out malignancy. The patient received the following antiluetic treatment during his stay in this hospital from September 26, 1932 to February 7, 1933: Hypodermic injection of bismuth every three days for 23 days; potassium iodide, 15 drops three times a day, increased gradually to 109 drops three times a day, the dose the patient was taking on date of his discharge.

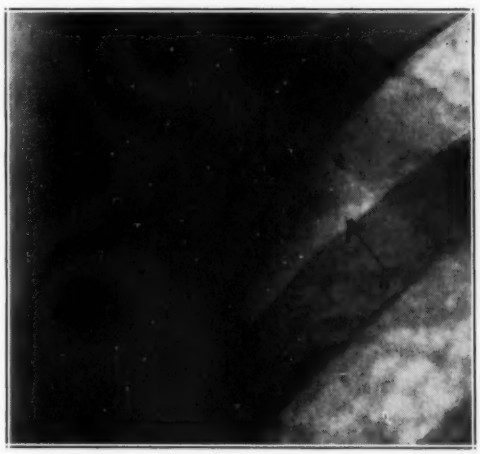

FIGURE 1.

Final discharge note states: "Patient is feeling quite well and has gained 35 pounds."

Skiagram of November 11, 1932 (fig. 2) showed complete healing.

FIGURE 2.

SUMMARY

It is my opinion that the case described is undoubtedly one of fracture of the eighth rib due to gumma, a very unusual finding nowadays.

NEW ORLEANS
Medical and Surgical Journal

Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

Clarence A. Lorio, M. D., Ex-officio

For one year: Leon J. Menville, M. D., Acting Chairman.
William H. Perkins, M. D.
For two years: Lucien A. LeDoux, M. D.
H. W. Kostmayer, M. D.
For three years: John A. Lanford, M. D., Secretary.

EDITORIAL STAFF

John H. Musser, M. D.....*Editor-in-Chief*
Willard R. Wirth, M. D.....*Editor*
G. C. Anderson, M. D.....*Associate Editor*

COLLABORATORS—COUNCILORS

H. E. Bernadas, M. D.
Daniel N. Silverman, M. D.
C. C. deGravelles, M. D.
M. D. Hargrove, M. D.
J. B. Vaughan, M. D.
Rhett McMahon, M. D.
C. A. Martin, M. D.
W. F. Couvillion, M. D.

Paul T. Talbot, M. D.....*General Manager*
1430 Tulane Avenue

SUBSCRIPTION TERMS: $3.00 per year in advance, postage paid, for the United States; $3.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the twentieth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

Manuscripts should be addressed to the Editor, 1430 Tulane Avenue, New Orleans, La.

MEDICAL ECONOMICS

One among the many unsubstantiated and wild statements that have been made by the proponents of the Wagner Bill has to do with the need of medical care by a large proportion of the population of this country. It has been stated that there are forty million people who are suffering from lack of medical care. The absolute unsoundness of this statement may be readily disputed by every practicing physician who comes in daily contact with rich and poor in his community. However, in order to disprove this statement which has been advanced *ex cathedra*, Dr. Brash, of Rochester, was appointed chairman of a committee by the Trustees of the American Medical Association to investigate the true facts relative to the need and supply of medical care in the United States. This committee reported to the House of Delegates of the American Medical Association at the recent meeting in St. Louis. Their findings should be of interest to all medical men.

Carried out with the cooperation of county and state medical societies, the replies to questionnaires were received from some twenty thousand physicians and dentists. Seventeen thousand of these forms were made out by physicians and covered a section of the population represented by forty-three million people. The information obtained by the committee showed definitely that some 10 per cent of the population were given free services by the medical man, estimated, in a general way, to be very much over the $1,000,000 a day of free service supposedly given by physicians in the United States.

In addition to the professional man, nurses, hospitals, health departments and relief organizations and social service departments were contacted and questioned. "Ninety per cent of all the sources consulted reported that they knew of no significant number of persons needing or seeking medical care who are unable to obtain it." The majority of opinions from all sources show that it is forty thousand rather than forty million people in the United States who lack or need medical services. The questionnaires indicated with almost complete unanimity that medical services supplied by governmental agencies were the ones most likely to be insufficient.

A study has been recently completed by the Council on Medical Education and Hospitals of the need for medical care in an

essentially rural state, Mississippi. Here again a very careful survey indicates that there is no lack of medical care, or hospitalization if needed, for the general population of the state. The urban and rural populations are both adequately taken care of in every way.

Both of these two studies indicate definitely that the pouring out of untold millions for the care of those who are presumed not to be able to obtain medical services is a piece of horribly rank extravagance. Were this money to go to the physician and to those who work under him immediately, taking care of patients, it might be money well spent and would recompense the doctor for the immense amount of free service he now gives to the indigent in office, in home, in clinic and in hospital. Such would not be the case. The bulk of the money would probably go to non-medical officials and for purposes not directly devoted to medical care. All those who come in personal contact with the sick now realize and appreciate that, irrespective of a man's financial status, efficient medical care may be obtained for him. To destroy the practice of medicine in order to obtain an objective which is chimerical and visionary would seem the height of political, social and economic folly.

THE VITAMINS

This is a subject which has been discussed very frequently in these columns in the last several years. However, it is still a very live subject and one about which an enormous number of papers are appearing in the current medical literature. In the last six months in the Quarterly Cumulative Index Medicus are 693 references to the subject of vitamins. It is not only the internist but the specialist as well who is interested in the expressions of vitamin deficiency and their eradication. Illustrative of this very general trend of the use of vitamins in the treatment of disease is the fact in the last two years there has been something like 1000 per cent increase in the sale of vitamin concentrates by the pharmaceutical houses. Vitamins have been found to correct many unusual conditions.

Of interest may be a few of the recent uses of vitamins which have not been generally known. For example, vitamin A is being used extensively by the ophthalmologist in the prevention of various types of ocular disease, notably indolent ulcers and chorioretinal disturbances. The dermatologist is using this vitamin to correct an eruption which is described as follicular and which resembles goose flesh and observed in those who have obtained sexual maturity. The surgeon has found that vitamin A is an excellent application for the treatment of burns and wounds, and that healing is accelerated by giving cod liver oil by mouth and using the vitamin locally. The urologist is using vitamin A in the treatment and prevention of urinary lithiasis. Some reports as to chronic vitamin A deficiency playing a role in the etiology of bladder and kidney stones are contradictory but the evidence seems to be accruing that a minimal vitamin A deficiency may play a part in producing these stones. The gynecologist has found that vaginitis, notably in elderly women, is materially benefited by giving a large amount of vitamin A.

Vitamin B, and more particularly that fraction which is now recognized as nicotinic acid, has proved of inestimable value in the management of patients who are suffering from subclinical pellagra. Patients who complain of nervousness, abdominal pain, intermittent diarrhea, weakness and insomnia, have improved remarkably by the administration of 50 milligrams of pure synthetized vitamin B_6.

Vitamin C has comparatively recently been shown to be of considerable help in the management of patients with anemia. It will accelerate the action of insulin so that the diabetic should be given large amounts of natural vitamin C. Healing seems to take place more rapidly in the patient who has a gastric ulcer when vitamin C, which is so often omitted in the ulcer diet, is administered in large doses.

It has been shown that the tuberculous individual requires a greater amount of ascorbic acid than does the normal. A small child who has diarrhea, a condition in which vitamin C is usually reduced, improves much more rapidly when vitamin C is added to excess in the diet than when it is omitted. Reports are coming in of improvement in patients with rheumatoid type of arthritis when given this preparation. It has been shown that it is an important factor in preventing anaphylaxis or allergy. As vitamin C is frequently withdrawn from the diet, caution should be observed in doing this. If it is necessary to take away the natural substances rich in vitamin C then it is advisable to give the concentrated tablets in 10 to 50 milligram doses two or three times a day.

Vitamin D, suffice it to say here, is essential to be given in adequate quantities in order to promote utilization of phosphorus in subclinical rickets.

Vitamin E is abundant in nature but this so-called antisterility vitamin has been given to women who abort habitually, or who are sterile, with some evidence of benefit.

Vitamin K, the antihemorrhagic factor, has proved of great value in the treatment of hemorrhagic states and more particularly has it been shown to be of value prophylactically prior to operation.

Vitamin P, with vitamin C, helps regulate vascular permeability. It has not as yet been used except experimentally.

Vitamin M is another vitamin which is of scientific or experimental interest but as yet lacks clinical utilization. This hypothetical vitamin, when absent from the diet, will produce severe stomatitis and granulocytopenia in the experimental animal.

Mentioned above are just a few of the more recent observations on the use of vitamins. Vitaminology may be a present day fad and the extensive use of vitamins as nowadays practiced may be, in the future, materially reduced. In the present transitory phase from the experimental to the clinical use of various vitamin preparations an immense amount of information is accruing to medical knowledge. Ultimately vitamins may not be so generally prescribed as at present—but in certain conditions and disease states they will be added to the accepted remedies of worth and value.

TO SECRETARIES OF PARISH SOCIETIES

The Journal Committee and the Editor have been very anxious indeed to increase the amount of space given to current activities amongst the various Parish and District Medical Societies. The Editor has addressed a personal note to, and by word of mouth in some instances, the secretaries of every medical society in the state. He has almost beseeched those who have secretarial duties to send in to the Journal office the information concerning the activities of physicians of his parish or of his district and information regarding medical meetings. In spite of diligent efforts the responses to those appeals have been answered by but a few.

Again the Editor, in behalf of the Journal Committee, requests that the state secretaries cooperate with him in order to make the New Orleans Medical and Surgical Journal livelier than it now is and one that represents more fully than it does the medical profession of the state. Doctors are interested in what other doctors are doing; they are interested in the programs of medical societies. As this news, for the most part, can only be obtained by publishing it in the Journal, which reaches most of the physicians of the state, the Editor then is appealing again to the secretaries to send in to the Journal news of their organizations. At least do this—please put the Journal on the mailing list for all programs and announcements that are sent out. Even this would be of some value because it would result in the publication of the notices or programs that are emanating from many sources throughout the state.

HOSPITAL STAFF TRANSACTIONS AND CLINICAL MEETINGS

TRI-STATE HOSPITAL, INC.
Shreveport

The regular monthly meeting of the Tri-State Hospital Staff was held May 11, 1939, at 8:00 p. m. with 23 members present. Dr. T. J. Bush called the meeting to order. Minutes of the previous meeting were read and approved without correction.

Dr. W. B. Allums presented the case of a child (884-L), aged 3, who had suffered a small rectal hemorrhage. There was a history of gonorrheal vaginitis, not proved bacteriologically. The child was very nervous and had eneuresis. Rectal examination revealed a foreign body apparently present in the vagina. When the vagina was probed the wire loop struck a metallic object. The x-ray revealed three safety pins in the vagina. These were removed under gas anesthesia and child recovered from all symptoms. The case was discussed by Drs. Wolfe and Strain.

Dr. J. E. Knighton, Sr., presented the case of a white male (1122-L4), aged 48, who was first admitted to the hospital on April 2, 1939 complaining of lethargy, dyspnea on effort, swelling of feet and ankles. Thyroidectomy had been done elsewhere 12 years before. Patient had been on maintenance dose of thyroid for a long period but had had none within the past several years. The symptoms complained of had been progressive for past year. Examination revealed dull facies, obesity, hoarseness, and non-pitting edema. A basal metabolic reading was −68. There was a microcytic anemia. He was given 2 grains of thyroid three times daily and in two weeks showed remarkable improvement. During treatment he had marked diuresis and weight loss. Basal metabolic rate at this time was −2.

Four days later he returned complaining of epigastric distress. During that night he had acute retrosternal pain, with shock, collapse of pulse and blood pressure. Electrocardiogram showed definite evidence of myocardial infarction with complete auriculoventricular block. Patient did not respond to treatment and expired after about 30 hours.

Two similar cases of myxedema, which had been treated elsewhere as pernicious anemia, were reported. Drs. Hargrove and Wolfe discussed the cases.

The hospital statistical report was read and deaths of the previous month reported.

E. W. Booth, M. D., Sec.

SOUTHERN BAPTIST HOSPITAL
New Orleans

The regular monthly staff meeting of the Baptist Hospital was held on Tuesday, May 23, at 8:00 p. m. Dr. E. Z. Browne, Chairman, presided and Dr. Garland Walls was in the Secretary's chair.

Dr. E. McC. Connely and Dr. Gilbert Anderson presented a case of spinal cord tumor. Operation was performed one year ago, with removal of a dural endothelioma. The patient made a complete clinical recovery and is now at work. Drs. T. C. Clements and E. A. Ficklen also presented the case of a patient from whom a dural endothelioma had been removed. Patient made a clinical recovery.

The remainder of the scientific program was devoted to a symposium on therapy in pneumonia. Dr. L. C. Chamberlain discussed dehydration therapy; Dr. Sam Hobson the serum therapy and Dr. P. H. Jones the use of sulfapyridine. The papers presented were freely discussed, Dr. Ervin Nelson, Professor of Pharmacology of Tulane University, and Dr. Allan Eustis opening the discussion.

The meeting of the Staff of the hospital was held on Tuesday, June 27, 1939, at 8 p. m. The following program was presented:

"Summer Diarrheas and Case Report of Autobiliary Intraduodenal Feeding," by Dr. Daniel N. Silverman; "Most Prevalent Contact Dermatitides," by Dr. Barrett Kennedy; "Final Results of Devine Colostomy (Case Report)" by Dr. E. A. Ficklen; "Uterine Myoma and Pregnancy (Case Report)" by Dr. John Dicks.

Garland Walls, M. D., Sec.

J. T. NIX CLINIC
New Orleans

At the meeting held in June, Dr. Joseph A. La Nasa presented the following paper:

URETERAL CALCULI

The subject of calculi in urinary disorders has always commanded the utmost interest. Difficulties in diagnosis are rather common occurrences. Atypical symptomatology in the presence of urinary calculi is frequently encountered and the avoidance of mistakes in the differential study requires the utmost skill.

It is not within the scope of this paper to discuss the etiology of stones in the urinary tract. However, it is most likely that in the formation of stones there are varying causes, and that no one theory will explain all cases.

Infections in the urinary tract, vitamin deficiency, changes in the acidity of the urine, colloidal disturbances, disease of the parathyroid gland, and foci of infection are all, no doubt, factors in the formation of stones.

Since the scope of this paper is to deal with ureteral calculi, the problem arises as to whether these stones are primary or secondary in the ure-

ter. The chief question of clinical interest here, is whether calculi which are present in the ureter, originate there, i. e., are primary or whether they simply represent the migration towards the bladder, of calculi which are formed in the kidney, that is to say, are secondary in the ureter.

The opinion of the majority of urologists is that, in nearly every case, the ureteral calculus was formed in the kidney and is found secondarily in the ureter. The minority, however, believe that calculi are formed primarily in the ureter as a result of the slowing up and subsequent infection of the column of urine, above a strictured area. Although one cannot deny that such a mode of formation holds true for a certain small percentage of cases, it seems illogical to ascribe such a primary origin to all cases, especially when there is an absence of any stricture.

As a rule there is only a single calculus, but there are many instances in which multiple calculi are found. Bilateral occurrence however, is comparatively uncommon. A large variety of combinations may be encountered:

(1) Ureteral and renal on the same side.
(2) Renal on one and ureteral on the opposite side.
(3) Bilateral renal and unilateral ureteral.
(4) Bilateral renal and ureteral, and also a vesical calculus.
(5) Bilateral ureteral and unilateral renal.

One must never fail to take these various possible combinations into consideration before deciding on which method of treatment is to be taken because of the danger of postoperative anuria.

The surface, composition and form of ureteral calculi resemble somewhat those of renal calculi. The surface is usually smooth or finely granular but may be quite rough, so that the calculus is easily caught in the folds of the epithelial lining of the ureter. The composition varies like that of renal calculi, with the exception that a relatively larger proportion are of the pure uric acid and urate variety and are therefore, often permeable to the x-ray.

The diagnosis of ureteral calculi is based upon a carefully taken clinical history and the results of a thorough urologic examination. The urologic examination should include besides a complete urinalysis, studies made by radiography, cystoscopy and ureteropyelography.

In the treatment of ureteral calculi, the trend in the last few years has been towards non-operative measures instituted to facilitate the passage of these stones into the bladder. It has been found that delivery of the calculus without operation is possible in three out of four cases. There has been much discussion as to the maximum size that may be manipulated safely. Opinions vary, but the prevalent one is that stones larger than 0.5-0.75 cm. should be removed by ureterolithotomy rather than by transureteral manipulation. It also has been agreed rather generally that only those stones which are situated in the lower third are suitable for manipulation. With regard to size of stones, the deciding factor is not the size of the stone but rather the diameter of the ureter and that no stone should be manipulated if there is not sufficient room in the ureter. One method of deciding this point is by preliminary passing of catheters alongside the stone. It should be possible to pass two No. 5 ureteral catheters easily beyond the stone before passage of extractor is attempted. When the extractor is passed it should never be forced. If these factors are kept in mind, a minimum of difficulty will be encountered.

Even though these rules are adhered to, there is a possibility that stones may become arrested somewhat in the course of withdrawal. This is rather uncommon, but it happens often enough to be important. One must be careful not to exert too much traction, or injury to the ureter may occur. Such a situation presents a real problem to the surgeon and may necessitate ureterolithotomy.

In conclusion I wish to stress strongly the point that in the large majority of cases of ureteral calculi that come to the attention of the urologist the stone can be dislodged by cystoscopic maneuvers. This condition occurs in three out of every four cases.

HUTCHINSON MEMORIAL CLINIC

New Orleans

Scientific Session conducted by the Department of Gynecology, Dr. E. H. Countiss presiding.

Theories as to the Cause of Primary Dysmenorrhea (Dr. E. H. Countiss): Ehrenfest's summary of work done on dysmenorrhea from 1933 to 1936, inclusive, will be quoted extensively. He states that "the profound anatomic, functional and biochemical changes, which during her child-bearing age periodically prepare the woman for a possible pregnancy, necessarily will give rise to a variety of subjective symptoms of which some express themselves in a marked pelvic discomfort."

Goldschmidt, in 1935, concluded that the disagreeable phenomena of menstruation are chiefly mental and traditional rather than biologic and that "women are still under the spell of the primitive taboo." Mayer restates the known fact that dysmenorrhea develops on a neurotic basis and may represent solely a protest or fear reaction, an "escape into disease."

Although dysmenorrhea does not necessarily go with sterility, it does indicate a causative relation in many instances because of the fact that the sterile patient is likely to overemphasize any slight anomaly of menstrual function because in her belief it is significant in inability to become pregnant. Until ten or fifteen years ago, most theories of menstruation were purely on a mechanical basis. However, it is now known that intrinsic or primary

dysmenorrhea requires careful clinical study, occasionally implying physical, biochemic, endocrinologic and psychologic examinations.

We may recall many dysmenorrheic patients with lumbar lordosis, thoracic kyphosis, visceroptosis, these being associated with pelvic congestion at the time of menstruation. Industrial girls may overemphasize their complaints in order to gain time off, particularly if their employers assume that menstruating women should be semi-invalids. Fekete states that even in an infantile uterus, menstrual pain is not due to cervical stenosis but more likely to compression of engorged endometrium within the rigid walls of the uterus, and he states that dilatation and curettage is only temporary by virtue of the fact that it causes the uterus to become softened by the hyperemization. It seems that diathermy or follicular hormone accomplishes the same purpose. About 50 per cent who undergo dilatation and curettage derive some benefit.

Tedstrom and Wilson found that in women who present blood sugars of 80 mg., pain could be relieved in 80 per cent by a high carbohydrate diet for a week before menstruation. Altschul found complete relief in four patients within thirty to sixty minutes after the administration of orange juice and insulin, and also found relief in some by the administration of a high carbohydrate diet. Reynolds, in 1932, noted that the rabbit uterus in rhythmic activity was dependent on estrogenic hormones of the ovaries and that this was inhibited by the corpus luteum. Witherspoon was an ardent supporter of dysmenorrhea due to deficiency of corpus luteum. Cannon, in 1936, states that the dysmenorrheic pain is essentially spasmodic or tetanic in character and it is generally agreed that all types of visceral pain are due to exaggerated muscular contractions. Whitehouse, in 1926, claimed that any abnormal detachment of menstrual decidua is invariably associated with pain. Cannon concedes that decidual tissue acts as a foreign body, provoking muscular contractions. Pohl stopped a so-called dysmenorrhea by the administration of typhoid extract. Clow, in 1932, stated that she had very little use for drugs in essential dysmenorrhea, and urges for patient to carry on daily routine as regards diet, bathing, exercise and work.

Again we come to the theory of Reynolds and Novak, based on a study in which they found that estrin stimulates uterine contractions, that is, the corpus luteum inhibiting factor. Israel and Cannon state that such a hormonic deficiency would have to manifest itself in a deficient formation of endometrium. Mazer found that 14 out of 20 patients with dysmenorrhea have normal endometrial progestin phase. According to Israel, a neurogenic basis has been advanced by some authors who ascribe the etiology of primary dysmenorrhea to an abnormal reactivity of the cervical nerves, leading to spasm of the circular musculature. On this basis, superior hypogastric plexus resection has been evolved. Davis, in 1936, blocked the Lee-Frankenhäuser plexus by means of 1 c.c. of 95 per cent alcohol and cured six, but she states that it was not without danger. Long recognized that improvement follows administration of thyroid in selected cases. Hertzler and his school speak of 'goiterous dysmenorrhea,' which they attribute to an interstitial thyrotoxicosis. Fluhmann reports a normal peak of estrogen concentration in the blood from eight to fifteen days before menstruation, with a frequent secondary rise just before, or at the time, of the menses. In 19 dysmenorrheic patients, blood estrogen failed to show any consistent departure from normal, either in amount or type of curve. He states that there are three categories of theories of cause of dysmenorrhea which are based on disturbances of ovarian hormones: (1) Deficiency of estrogen; (2) an excess of estrogen or a deficiency of progestin; (3) overactivity of progestin.

As to the deficiency of estrogen, the association of dysmenorrhea with hypoplasia uteri, oligomenorrhea and hypomenorrhea early led to the belief that it may be due to hypoovarian condition. Castration produces degenerative changes in the ganglia of Frankenhäuser, which may be corrected by estrogen administration. Consequently, it was suggested that a lack of sufficient estrogen causes disturbances which affect the sympathetic nerve supply of the uterus, whereupon dysmenorrhea ensues. This is not compatible with two observations: (1) Dysmenorrheic women do not show a diminished estrogen; (2) existence of a hypoplastic uterus as evidence of ovarian deficiency is demonstrable in a limited number of cases only.

As to the excess of estrin or progestin deficiency, estrin stimulates uterine contractions. It was found that progressive muscular activity during the estrin phase was quite pronounced, while in the postovulatory or progestin stage it is quiescent. The objections to this theory were: (a) An overactivity of estrogenic hormone uninhibited by progestin is completely fulfilled in hyperplasia endometrii, but the bleeding in this condition is almost always painless; (b) hyperestrinism in the blood at the time of menstruation is not more frequent than in women without dysmenorrhea. However, Katz and Parker, using the Frankenhäuser-Goldberger test, found a higher level of estrogenic substances in the blood of a large percentage of dysmenorrheic patients; (c) the peak of estrin and uterine contractions is reached at the time of ovulation, but except for women with 'Mittelschmerz' dysmenorrhea, patients do not have pain at this time; (d) a deficiency of corpus luteum is difficult to understand as a cause since there is normally a deficiency at this time. (It undergoes degeneration two or three days before menstruation and lack of progestin is indicated by absence of pregnanediol in urine).

As to the excess of progestin, Cannon assumes that dysmenorrhea resulting from macroscopic separation of endometrial fragments instead of the usual molecular degeneration is fundamentally due to overactivity of corpus luteum hormone, which is in turn due to overactivity of the luteinizing hormone of the anterior pituitary body. However, progestin disappears one or two days before menstruation and excessive doses do not lead to abnormal desquamation of the endometrium.

Schwarz and Smith divide dysmenorrhea into three classes: An organic form, depending on obstruction, a congestive and a neuralgic form. Schroeder thinks that there is an excessive irritation of the vegetative nervous system by toxic substances, evoked by the function of menstruation, and Graves suggests that this theory is supported by the relief secured from the administration of atropine. It has also been shown that the application of cocaine to the tuberculum septi, designated as the genital spot of the nose, has relieved dysmenorrhea. Schwarz reported 12 cases with complete relief in eight and partial in four after eliminating causative agents from the diet.

With these theories, we are left many on which to work, none of which are applicable to all cases. I heartily agree with Clow of England who, in a study of 15,000 women, allowed the patients to continue their daily tepid baths and postural exercises, and had less dysmenorrhea after a year of such treatment. I believe that we will accomplish more by starting earlier on these patients. We believe in some, at least, that there is a congestion within the pelvis prior to and during menstruation which blocks the cervical canal and probably accounts for some cases of dysmenorrhea.

The Conservative Management of Dysmenorrhea (Dr. R. E. Rougelot): Since speculation as to the etiology of dysmenorrhea is still rife, the well-known medical axiom that the number of therapeutic methods in use should be legion is well borne out.

Beginning with the patent medicines such as vegetable compounds, long condemned but still used by some of the more gullible, the list of agents used runs through the pharmacopeia, including iodides, analgesics, antispasmodics, sedatives and narcotics, while other treatments include physiotherapy, psychotherapy, endocrine therapy, x-ray, insulin therapy, surgery, and various other therapies too numerous to mention, some related to medicine, others related to folk lore and superstition so closely attached to the menstrual and sexual life of woman.

To get back on firmer ground, we must remember that we are dealing with a symptom, like hypertension or angina, most likely part of a disease complex whose further ramifications may be revealed in the light of future study, and that the primary type known as essential dysmenorrhea, may at some time be retired into a category in relation to other conditions similar to that in which the present types of acquired or secondary dysmenorrhea are now placed. Inasmuch as the acquired type usually has its origin in some well-defined or evident pelvic pathology in which the menstrual period causes exacerbation with attendant pain calling for appropriate and indicated treatment, we propose in this paper to confine our remarks to the essential type, and, without too much discussion of etiology, carry over to a consideration of those conservative measures in use today which seem to give workable results in the relief of this troublesome condition. It is conceived that primary or essential dysmenorrhea is associated immediately with increased contractility of the uterus and perhaps a lowered threshold for pain, since normal women may show similar uterine contractions without experiencing pain. That muscular contraction causes pain is well borne out by recalling the severe suffering experienced in intestinal cramps or the discomfort of a cramped leg muscle. And indeed experimental workers such as Novak and Reynolds, Knaus, Lackner, and others, have concluded that there is a rather close connection between exaggerated uterine contractility and primary dysmenorrhea. The presence of a hypoplastic uterus, a spastic cervix, or a congested endometrium, does not obviate the fact of increased contractility, but helps explain why the uterus exerts itself in an effort to overcome these conditions.

Moir suggested from experimental observation in which he noticed that arterial pulsation was absent during uterine contraction, that the pain might be associated with ischemia of the uterus similar to the heart in angina pectoris, or the skeletal muscle in intermittent claudication; while Lackner, in this country, has not been able to substantiate this, and Elden and Wilson feel that some of the pain may be due to a lowered sympathetic threshold.

Dysmenorrhea, literally - difficult - monthly-flow, has an incidence ranging from 22 to 50 per cent. It is not a regular monthly occurrence and is not necessarily concomitant with the menarche, a point on which many claim a psychogenic origin. Symptoms are not confined to the uterus but may include headache, nervousness, anorexia, nausea and even diarrhea. Pain is confined to the day or so before the flow and is usually relieved when the flow is established. This time corresponds with the point at which corpus luteum influence is on the wane, and is the basis for progesterone therapy.

The treatment of the immediate attack of dysmenorrhea is symptomatic, and usually suffices. Of the various analgesics, aspirin or a combination of acetanilid and acetphenetidin, will give good results. Viburnum has long enjoyed a reputation as antidysmenorrheic, and Parke-Davis' Liquor Sedans, of which hydrastis is the chief ingredient, has wide use. Perhaps the alcoholic content of many patent medicines is the most active ingredient, instead of the well-advertised herbs.

Iodides are recommended by some, and calcium therapy has taken the fancy of others for its reputed relaxing effect on muscle. Antispasmodics have enjoyed a wide use among which benzyl benzoate, hyoscyamus and atropine are to be noted. The latter, most often employed as the tincture of belladonna, is given in doses of ten drops every four hours, either alone or in combination with some sedative, such as phenobarbital. At times, codeine is necessary in one-fourth or one-half grain doses, but only for severe cases. It has been my experience that nurses use codeine more than any other group, probably because of its availability. Morphine is used occasionally, but not without reluctance. A common prescription said to give good results, at Stephens' College, Missouri, consists of: Camphor monobromate gr. ss; atropine sulphate gr. 1/500; papaverine hydrochloride gr. ¼; acetphenetidin; acetyl salicylic acid aa gr. iii; M et ft caps as above.

After the symptoms subside, one must attempt to obtain permanent relief of the underlying condition. Every patient should have the benefit of a thorough history and physical examination, including the necessary laboratory study and a basal metabolism test if feasible. Occasionally, uterosalpingography is done and every effort must be made to be sure that one is dealing with a functional type and that no underlying pathology is overlooked. The patient's general health must not be neglected. Attention to bowels, proper mental relaxation, avoidance of fatigue, proper hygiene, correction of poor posture, clearing up a secondary anemia, a balanced diet usually high in vitamins, a change in climate, systematic exercise, and out-of-door activities all promote the patient's well being and improve the nervous and mental makeup. German investigators take the radical view that functional dysmenorrhea is of psychic origin and treat it accordingly, and Mathieu feels that many patients require a psychiatrist. He states that all young girls approach the menarche with fear induced by what they have been told about menstruation and by the way they have been prepared for it. There is no doubt that many women have a wrong attitude towards menstruation, and that pain or a consciousness of subthreshold feelings is exaggerated, or perhaps the period is used as a defense mechanism to escape some unpleasant situation. Consequently, one should evaluate the nervous background of his patients in order to eliminate or treat this factor. There is still a great deal of controversy as to the ill or beneficial results of exercise. The results obtained with this method of treatment are surprisingly good. Alice Clow, at a girls' school in England, reports that the proper mental attitude coupled with outdoor activities, such as competitive sports and a daily hot bath, has reduced dysmenorrhea in 75 per cent of hundreds of her cases among young women and girls.

Brown has adopted a similar procedure at a girls' school over here, with good results. The assumption is that exercise increases circulation, thereby allaying nerve irritation and improving nutrition. Other investigators in Japan and Italy take the opposite view that exercise is not beneficial and instead of relieving pain increases the flow while such activities as swimming may actually check it due to the chilling of the body.

Occasionally, one is tempted to treat the patient on an allergic basis. Schwarz and Smith report on five women with food sensitivity and dysmenorrhea which was relieved by elimination diet. However, Tuft states that it is the exception rather than the rule and perhaps this is substantiated to some extent by the fact that food allergy is present in a large proportion of women who do not suffer particularly from dysmenorrhea. At times insulin has been used with good results, the rationale being that it improves the uterine metabolism, but this treatment is largely discontinued. With the development of endocrines, it was but natural that they should be tried on this condition which has been suspected of being of endocrine origin for many years.

Fluhmann, in a study of 19 women with primary dysmenorrhea, found that the blood estrogen level showed no consistent departure from normal either in the amount of estrogen present, or in the type of curves obtained, and concluded that there are serious objections to the acceptance of any theories which seek to explain the cause of primary dysmenorrhea on the basis of a deficiency or excess of either estrogen or progestin.

It may be well to state here that oral therapy, with the various preparations which glut the market, is unwarranted both from a consideration of their efficiency and from the expense involved. When estrogenic hormones became available, it was but natural that it should be tried on dysmenorrhea, and indeed many reports are still favoring its use. However, in the light of our present knowledge that estrogenic hormone actually sensitizes the uterus to pituitary extract, thereby rendering the muscle more contractile, there seems but little indication for its use except where there is a hypoplasia of the uterus. We therefore attempt, by substitution therapy, to stimulate uterine growth and, when used, such preparations as theelin, amniotin, emmenin, or progynon B, are administered hypodermically in doses of 10,000 units, twice a week for several months, while the uterus is checked at intervals for increase in size. Where there is evidence of hypothyroid activity, the desiccated gland is given in appropriate dosage in proportion to the lowered metabolism. Of all the treatments for dysmenorrhea, however, progesterone therapy is coming into its own. There is sound reasoning for its use, based on careful experimental work, and apparently better results are obtained with this hormone than any of the others.

Its use is predicated on the fact that corpus luteum definitely lessens uterine contractility. Elden and Wilson were able to give complete relief to 47 per cent of their patients and 11.7 per cent partial relief, using dosages of progesterone ranging from 2/25 to 1 rabbit unit, given three to six days before the menses, in single or divided doses. It is noteworthy that there was no effect on the character or duration of the menstrual period. These results have been confirmed by others including careful experiments with intrauterine balloon, and kymograph tracings by Lackner, Soskin and Krohn. Our department is also using progesterone in doses usually of one-half to one rabbit unit daily, beginning about three or four days before menstruation, and continuing into the period. It must be remembered that this type of therapy is at best temporary, and substitutional in nature.

Since the development of the masculine hormone, testosterone proprionate, gynecologists have used it because of its similarity, chemically, to progesterone. Robinstein and Arbarbanel found antuitrin S disappointing and experimented with testosterone because it depresses pituitary gonadotropic secretion and inhibits ovulation, leading to a suppression of estrogen formation and, in addition, favors luteinization and maintenance of the corpus luteum increasing its progesterone effect, leading to myometrial relaxation, myometrial growth through its trophic effect and progesteronal response in the endometrium.

In my opinion, however, testosterone has no place in this disease since it does depress pituitary gonadotropic secretion and inhibits ovulation and because we do not know what the ultimate effect of a masculinizing hormone might be in the young women so treated. Of late, x-ray irradiation of the pituitary has been used with excellent results obtained by Katz and Parker, using it either alone or in combination with gonadotropic luteinizing substance.

We may conclude then that the treatment of dysmenorrhea is still an open question subject to much debate and experimentation on a psychologic, physiologic or endocrine viewpoint but that of late accumulated evidence points favorably to its being of endocrine origin, while the ultimate solution in the light of intense modern scientific study rests perhaps not too far in the future.

The Surgical Treatment of Dysmenorrhea with Especial Reference to the Anatomy and Surgery of the Presacral Nerve (Dr. B. B. Weinstein): The cerebral fecundity of the theorist on causes of dysmenorrhea has only been rivaled by the prolific suggestions of the therapist. I deeply appreciate Dr. Countiss' and Dr. Rougelot's fine expositions which relieve me of the necessity of reviewing this material. Suffice it to say when the general practitioner and the internist finally yield their patients to the gynecologist and when the gynecologist finally selects some method of surgical therapy, all of the simpler modes of treatment have been exhausted.

Dysmenorrheas may be divided into primary and secondary types. The secondary type is incident to some demonstrable pelvic pathology and when the pathology is adequately dealt with the dysmenorrhea abates. In the time allotted, it would be impossible to review the now standard surgical procedure for the correction of displacements of the uterus, cervical stenosis, uterine leiomyomata, and endometriosis which are the lesions commonly associated with secondary dysmenorrhea. The surgical therapy of secondary dysmenorrhea will therefore be dismissed with the comment that correction of the underlying pathology in most cases effectively relieves the dysmenorrhea.

Essential, idiopathic, or primary dysmenorrhea may occur in association with organic pelvic pathology, but is not secondary to it, and frequently exists independently.

Our surgical armamentarium as applied to primary dysmenorrhea is rather limited. The simplest procedure is dilatation and curettage. An appreciable number of cases are benefited by this alone. In another group of cases dilatation and curettage followed by insertion of a stem pessary will give relief. When these procedures have been tried, and the dysmenorrhea persists, with the pain referred to the rectum, coccyx, sacrum and anus, the case is one in which some form of intervention on the sympathetics may be profitably tried.

A variety of procedures on the sympathetics have been suggested since Jaboulay in 1898 first advocated posterior approach to the pelvic sympathetics. The following year Ruggi suggested the transabdominal approach with resection of the ovario-uterine plexus. Neither of these suggestions was favorably received and in 1913 Leriche began his work on periarterial sympathectomy of the hypogastrics. Its publication in 1921 was followed by successful reports, but the difficulty of procedure was a deterrant to its universal applicability, and in 1925 Cotte suggested the simpler resection of the superior hypogastric plexus which procedure was seized upon avidly and followed by enthusiastic reports.

As Cotte's operation was widely practiced there developed a demand for an investigation of the anatomy of the hypogastric plexus. The early work had been done by Frankenhäuser in 1867. Later came the reports of Leriche, Latarjet and Bonnet, and more recently the anatomy has been investigated by Roussel, Delmas and Lux, Hovelacque, Cotte, Dovrzaniecki and Serafin, Elaut, Davis and Labate, and many others. The report this evening will briefly consider the work of the above, and material selected from 70 dissections made by me during the past year.

Investigators soon described various patterns assumed by the superior hypogastric plexus, though earlier workers had described single nerves

and bilateral cords. Elaut described four plexus types as well as cords and single nerves. Dovrzaniecki and Serafin describe four, while Labate has described seven patterns, all of which types I have found and in addition one more plexus type which occurs with some frequency, and has not heretofore been described. (The anatomy of the plexus and the plexus patterns were then described together with a lantern slide demonstration of the common types). As to the incidence of the plexuses, bilateral cords and single nerves, the following percentages have been recorded by various workers:

	Plexus per cent	Bilateral cords per cent	Single nerves per cent
Labate	84	8	8
Roussel	25	0	75
Delmas and Lux	80	0	20
Elaut	60	26	16
Kalberg	23	8	7
Learmonth	80	0	20
Davis	76	0	24
Our cases	88	10	2

Bergier, Morrison, Lacombe, single nerves only.

Cordier, Jianier, Bernard, Hartman-Weinberg, found plexus dominant as did Segond and Hovelacque.

In resection of the superior hypogastric plexus the midline incision seems preferable, the viscera are packed away and the interiliac trigone exposed. The peritoneum is excised and reflected and the nerve fibers identified. The entire fibrocellular tissue of the inter-iliac trigone is then removed. Extreme care should be utilized in the dissection because of the danger of injury to the middle sacral artery or to aberrant vessels or structures sometimes present in this area. The results reported have been quite good: Cotte (1929) 200 cases, 95 per cent relieved; Michon, 22 cases good results; Fontaine and Herrmann, 13 relieved out of 15; Adson, six successful cases; Counsellar and Craig, 14 cases—nine 100 per cent, two—95 per cent, three—75 per cent relieved; Abbott, eight successful cases; Donaldson, 15 successful out of 16; Graffagnino, six successful cases. There were many other reports in the literature which I have abstracted but have not time to present here.

In a thin subject with a shallow pelvis the procedure is extremely simple, but it is sometimes quite difficult in an obese individual with a deep pelvis. However, most of the difficulties with the procedure are concerned with anatomic anomalies. Davis reports three cases and I have seen another of aberrant ureter. There is frequently an anomalous vein running from the left common iliac vein to the pelvic wall, which may be easily injured. Injury to the middle sacral artery is frequent, and occasionally it is mistaken for a "presacral nerve," and ligated. The deviation of the left iliac vein medially causes it to project into the trigone where its fragile walls make it susceptible to damage on rough or careless manipulation. I have seen two cases of ptosis of the duodenum where the transverse duodenum crosses the interiliac trigone.

Another fact of importance is the position and relation of the sigmoid mesocolon. There are three chief types of mesosigmoidal attachment; the short mesocolon well out of the way of the field of operation which gives no difficulty; the long mesocolon which can be retracted from the field, and the long mesocolon, the leaves of which embrace the operative area. The latter type presents some technical difficulties in that one must dissect between the layers of the mesocolon to remove the plexus. This type has been found in one per cent by Labate, 15 per cent by Roussel, 8 per cent by Elaut, one per cent by Cotte, and we have seen it in 6 per cent. A note of caution in regard to dissection between the leaves of the mesocolon. The incision should be parallel to the root of the mesocolon in order to avoid extensive dissection which obscures anatomic landmarks and increases the likelihood of injury to the presacral artery (Elaut, Davis, Labate). Occasionally, the ureter has been injured. The left ureter is usually outside of the operative field. The right ureter should be identified since it crosses the field, transversing the right common iliac artery at the level of the lower border of the first sacral vertebra 3.5 cm. from the midline. Its position corresponds to the point of bifurcation of the common iliac artery. However, the right ureter adheres to the peritoneum and may be retracted with it, while the fibers of the superior hypogastric plexus remain adherent to the subjacent fibrocellular tissue.

When these anatomic anomalies are borne in mind, there remain two common difficulties, one of which is hemorrhage, chiefly from the middle sacral artery as a result of forceful severance from it, and the other is ligature of nerves resulting in neurofibromata, which have been reported several times.

Occasional failures have been reported. The foremost cause of failure seems to be improper selection of cases. I am convinced that cases are selected which have ovarian rather than superior hypogastric plexus pain. It has now been well demonstrated that the ovarian and hypogastric plexus are quite independent. This mistake can be avoided by the diagnostic lumbar block advocated by Flothow. Another cause of failure is the inadequate resection of nerve tissue. It is difficult to determine the presence of nerves by palpation, though they are visually obvious. Again, poor results are obtained because of the careless ligation of nerve fibers, with the later occurrence of neurofibromata.

Prior to sympathectomy, novocaine block may be used as a diagnostic procedure. Cassary and Bailey have reported permanent relief in one case follow-

ing diagnostic novocaine injection. Davis has recommended but has not used alcoholic block of the superior hypogastric plexus as a therapeutic procedure. Flothow boldly utilized this method and warmly advocates it. Alberta Davis reported six successful cases utilizing this procedure.

A final comment as to the use of the term "presacral nerve" which I wish to condemn strongly is, as has been demonstrated, that the superior hypogastric plexus is really rarely a single nerve, and is never presacral. It is usually a prelumbar plexus.

In conclusion, I may state the following: (1) There are a certain number of primary dysmenorrheas which may be relieved by dilatation and curettage or dilatation and curettage and stem pessary. (2) In a group of dysmenorrheas relieved in no other manner resection of the superior hypogastric plexus is a procedure which gives good results in adequate hands. (3) The anatomy of the region has been discussed with comments on seventy additional dissections. Stress has been placed on the variation in plexus patterns, and the anomalies of the region. (4) The utilization of novocaine block of the superior hypogastric plexus as a diagnostic aid, and of alcoholic block as a therapeutic measure, has been discussed.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR

July 3. Board of Directors, Orleans Parish Medical Society, 8 p. m.
Clinico-pathologic Conference, Hotel Dieu, 8:15 p. m.

July 5. Radio Talk, Orleans Parish Medical Society, WDSU, 8:15 to 8:30 a. m.
Clinico-pathologic Conference, Charity Hospital and L. S. U. Medical Center, 2 p. m.
Hutchinson Memorial Clinic Staff, 8 p. m.

July 6. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.

July 10. Orleans Parish Medical Society, 8 p. m. Second Quarterly Executive Meeting. Following this meeting the Society adjourns for summer recess.

July 12. Radio Talk, Orleans Parish Medical Society, WDSU, 8:15 to 8:30 a. m.
Clinico-pathologic Conference, Charity Hospital and L. S. U. Medical Center, 2 p. m.

July 13. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.

July 19. Radio Talk, Orleans Parish Medical Society, WDSU, 8:15 to 8:30 a. m.
Clinico-pathologic Conference, Charity Hospital and L. S. U. Medical Center, 2 p. m.

July 20. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.

July 26. Radio Talk, Orleans Parish Medical Society, WDSU, 8:15 to 8:30 a. m.
Clinico-pathologic Conference, Charity Hospital and L. S. U. Medical Center, 2 p. m.

July 27. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.

During the month of June, the Society held its regular scientific meeting and a special combined clinical meeting with the United States Marine Hospital.

At the meeting of June 8, the following program was presented:

The Relation of Histamine to Asthma and Urticaria.
By.....Dr. Allan Eustis.
Discussed by Drs. N. K. Edrington, J. H. Musser, F. F. Boyce, and closed by Dr. Eustis.

Use of Physiotherapy in Connection with Fractures and Injuries Involving the Joints.
By.....Dr. Guy A. Caldwell.
Discussed by Drs. R. H. Alldredge, N. H. Polmer and closed by Dr. Caldwell.

Hysterosalpingography.
By.....Dr. Thomas B. Sellers.
Discussed by Dr. P. J. Carter and closed by Dr. Sellers.

Final favorable action was taken on the amendment to Article 1, Section 2, of the By-Laws as follows:

"*ACTIVE MEMBERS*: Eligibility—Any reputable white physician residing in the Parishes of the First and Second Congressional Districts who is a natural-born or naturalized citizen of the United States and who is a graduate of a regular school of medicine and who is legally qualified under the medical law of Louisiana to practice medicine and surgery; also any reputable white physician who is a natural-born or naturalized citizen of the United States connected in an all time capacity with a recognized medical school or recognized approved institutional work of the First and Second Congressional Districts and who is not actively engaged in the practice of medicine."

The following program was presented at the clinical meeting at the United States Marine Hospital on June 26:

1. Hodgkin's Disease: An Experimental Study of the Etiology.
   By.....Dr. J. G. Pasternack.
2. Lymphogranuloma Inguinale: Treatment with Sulfanilamide and by Pyretotherapy.
   By.....Dr. J. A. Trautman.

General discussion followed.

Refreshments were served at the conclusion of the meeting.

Drs. Henry Leidenheimer, Jr., Hugh Page Newbill and B. Bernard Weinstein were elected to Active Membership.

Dr. Daniel N. Silverman attended the forty-second annual meeting of the American Gastroenterological Society at Atlantic City, May 1-2.

Dr. Wiley R. Buffington was a guest speaker before the Texas State Medical Association at San Antonio, May 8.

The following members attended the meeting of the American Psychiatric Association at St. Louis, May 8-12: Drs. Lewis A. Golden, Martha W. MacDonald, Walter J. Otis, Theodore A. Watters and Erwin Wexberg. Dr. Otis also attended the annual meeting of the Catholic Physicians' Guild in St. Louis.

Dr. Sydney Jacobs and Dr. William Harvey Perkins spoke at the 1939 meeting of the Council of Social Agencies on June 1.

Dr. Lewis A. Golden has been certified by the American Board of Neurology and Psychiatry.

Dr. Joseph C. Menendez gave the principal address at the Memorial Day services held at Biloxi on May 30, sponsored by the Veterans of Foreign Wars of the United States.

Dr. Rudolph Matas was awarded a certificate of honorary membership in the American Association of the History of Medicine at the sixth annual meeting of the History of Medicine Society of Tulane University. Faculty members presented honorary memberships in the local society were Drs. C. C. Bass, Elizabeth Bass, Maxwell Lapham, Ernest Carroll Faust, Isidore L. Robbins, E. Perry Thomas and Roy H. Turner.

Dr. Francis L. Jaubert was re-elected president of the Board of Directors of the Louisiana Society for Crippled Children at the May 27 meeting. Dr. H. Theodore Simon was among the speakers at this meeting.

Elected to the Board of Directors of the New Orleans Pure Milk Society are: Dr. Roy E. de la Houssaye, president, and Dr. E. A. Socola, vice-president.

Dr. Wilbur C. Smith has received a degree of Doctor of Science from Wake Forest College, North Carolina.

Dr. Béla Halpert read a paper before the National Gastroenterological Association in New York, June 1-2.

Dr. Dean H. Echols was elected to the Board of Directors of the University of Michigan Club of New Orleans.

Dr. Frederick Boyce announces the opening of his new offices at 1215 Union Building.

Drs. John S. Couret and Edwin H. Lawson announce the opening of their laboratory of clinical pathology at 952 Canal Bank Building.

Senior Surgeon William H. Slaughter will leave shortly to assume charge of the United States Marine Hospital at Chicago, and Past Assistant Surgeon John D. Lane, Jr., will come to New Orleans.

Dr. Guy A. Caldwell has been named Secretary of the Section on Orthopedic Surgery of the American Medical Association.

Dr. John H. Musser was re-elected a member of the American Board of Internal Medicine.

Drs. Allan Eustis, John H. Musser and Alton Ochsner attended the meeting of the Tennessee Valley Post Graduate Medical Assembly, Knoxville, Tennessee on June 28.

Miss Mary Louise Marshall attended the forty-first annual meeting of the Medical Library Association at the Academy of Medicine, Northern Newark, New Jersey, June 27-29. Miss Marshall is a member of the executive committee of this association.

Dr. Alton Ochsner addressed the Mississippi Dental Society, June 12, in Gulfport.

Drs. Eugene Countiss, Mims Gage and George Roeling attended the Tri-State County Medical Meeting at Hattiesburg.

TREASURER'S REPORT

Actual book balance 4/30/39	$4,952.64
May credits	$ 537.22
Total credits	$5,489.86
May expenditures	$ 853.08
Actual book balance 5/31/39	$4,636.78

LIBRARIAN'S REPORT

During May, 84 volumes have been added to the Library. Of these 54 were received by gift, 15 by binding and 15 from the New Orleans Medical and Surgical Journal. The Library has loaned to physicians during May, 1,089 books and journals, approximately two to each member of the Society. In addition 1,012 volumes have been loaned to students for overnight use, a total circulation of 2,101 for the month.

Members of the staff have collected material on the following subjects during May:

Obstruction and embolism of the retinal artery
Pyloric stenosis in infancy
Use of Vitamin B in multiple sclerosis
Painless hematuria
Intraspinous use of Vitamin B
Manic-depressive psychoses in identical twins
Medullary tumors of the adrenals
Sulfapyridine
Cryptococcus
Death from lipo-vaccine
Diseases due to Rickettsia
Perlèche

NEW BOOKS

White, W. A.: Twentieth Century Psychiatry, 1930.

Weihofen, Henry: Insanity as a Defense in Criminal Law, 1933.

Williams, F. E.: Adolescence, 1930.

Wolfe, W. B.: How to Be Happy Though Human, 1931.

White, J. C.: Autonomic Nervous System, 1935.

Practical Medicine Series: Eye, Ear, Nose and Throat, 1930. Dermatology-urology, 1931.

Scudder, C. I.: Treatment of Fractures, 1938.

Balyeat, R. M.: Allergic Diseases, 1938.

Pelouze, S.: Gonorrhea in the Male and Female, 1938.

Allen, Edgar, ed.: Sex and Internal Secretions, 1939.

Tidy, N. M.: Massage and Remedial Exercises, 1939.

Lapham, M. E.: Maternity Care in a Rural Community, 1938.

Marie, J. S. F.: English, German, French, Italian, Spanish Medical Vocabulary, 1939.

Crownhart, J. T.: Sickness Insurance in Europe, 1938.

Murphy, W. P.: Anemia in Practice, 1939.

Barsky, A. J.: Plastic Surgery, 1938.

Friendenwald, J.: Clinics on Secondary Gastric Disorder, 1938.

Blum, Sanford: Pediatric Symptomatology, 1938.

Stimson, B. B.: Manual of Fractures and Dislocations, 1939.

Davidson, F. R.: Manual of Toxicology, 1938.

Perkins, Harvey W.: Causes and Prevention of Disease, 1938.

Gilbert C. Anderson, M. D.,
Secretary.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge.
East and West Feliciana	First Wednesday in June	Jackson.
Morehouse	Second Tuesday of every month	Bastrop.
Orleans	Second Monday of every month	New Orleans.
Ouachita	First Thursday of every month	Monroe.
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	

A MINUTE OF THE JOURNAL COMMITTEE

It is felt that the death of the Chairman of the Journal Committee, Dr. William H. Seemann, will prove a great loss to the Committee and to the entire State Medical Society. Dr. Seemann had always been keenly interested in organized medicine and was very active in all of its undertakings. He graduated from Tulane University School of Medicine in 1900 and immediately affiliated with the Orleans Parish Medical Society and the Louisiana State Medical Society. He served as Treasurer of the Orleans Parish Medical Society in 1902 and 1903 and as President in 1909. In 1916-1917 he was President of the State Medical Society and represented this organization in the House of Delegates of the American Medical Association for more than twenty years. On April 11, 1929 he was elected as a member of the Journal Committee, serving as Chairman from May, 1935 until his death on November 19, 1938. His service to the Journal Committee will long be remembered and the result of his efforts will have a lasting effect on the future welfare of our Journal.

LAFOURCHE VALLEY MEDICAL SOCIETY

The Lafourche Valley Medical Society held its regular quarterly meeting at the Houma Country Club on June 21, 1939. The following program of scientific papers was presented: "Preventive Medicine: A Plea for Its Greater Popularization by the General Practitioner" by Dr. H. C. Dansereau, of Labadieville; "Treatment of Acute Disorders Due to Some of the Chemical and Physical Agents" by Dr. Louis A. Monte, of New Orleans; "Remediable Factors in Heart Disease" by Dr. Edgar Hull, of New Orleans.

Percy H. LeBlanc, M. D., Sec.

APPROACHING MEETINGS

The eighteenth annual scientific and clinical session of the American Congress of Physical Therapy will be held September 5-8, 1939 at the Hotel Pennsylvania, New York City. Preceding these sessions the Congress will conduct an intensive instruction seminar in physical therapy for physicians and technicians, August 30-September 2.

The ninth annual Convention of the Biological Photographic Association will be held September 14-16, at the Mellon Institute for Industrial Research, Pittsburgh, Pa. The program will be of interest to scientific photographers, scientists who use photography as an aid in their work, teachers in the biologic fields, technical experts and serious amateurs.

The University of Wisconsin Medical School is to conduct an Institute for the Consideration of the Blood and Blood-Forming Organs, September 4-6, 1939. The program is to include papers and round-table discussions by European and American workers in the field of hematology. Formal papers will be presented, among others, by Dr. L. J. Witts, Oxford, England, "Anemias Due to Iron Deficiency," Dr. E. Meulengracht, Copenhagen, Denmark, "Some Etiological Factors in Pernicious Anemia and Related Macrocytic Anemias," Dr. George R. Minot, Boston, "Anemias of Nutritional Deficiency;" Dr. Russell L. Haden, Cleveland, "The Nature of the Hemolytic Anemias"; Dr. Edward B. Krumbhaar, Philadelphia, "Hodgkin's Disease"; Prof. Hal Downey, Minneapolis, "Infectious Mononucleosis."

Physicians and others who are interested are cordially invited. A detailed program may be obtained by addressing Dr. Ovid O. Meyer, Chairman of Program Committee, University of Wisconsin Medical School, Madison, Wisconsin.

An International Course in Malariology, under the direction of Dr. G. Bastianelli, will be given in Rome, July 25-September 20, 1939. The program of this course may be consulted in the office of the Journal, or further information may be secured from G. G. Chiavari, Royal Consul of Italy, 4630 St. Charles Avenue, New Orleans.

The American Optical Company has recently issued a booklet entitled "The Ophthalmoscope and Studies of the Fundus Oculi in Important Pathological Conditions." A copy of this booklet may be secured without obligation by writing to the company at Southbridge, Massachusetts.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

At the recent meeting in St. Louis, 259 candidates were examined, 29 of whom failed. Future examination fees will be $75.00. The next examination, written for Group B, will be held Saturday, December 2, 1939. Applications must be on file in the secretary's office before October 4. The general oral examination will be held in Atlantic City, June 7-9, 1940.

A physician is needed in Cloutierville, Louisiana. For further information, call office of the Louisiana State Medical Society, Magnolia 3281.

HEALTH OF NEW ORLEANS

The Department of Commerce, Bureau of the Census, reports that for the week ending May 13, there were 140 deaths in the City of New Orleans. This was an increase of 24 over the previous week. Thirteen of the deaths were in infants under one year of age. The following week, closing May 20, found a slight increase in the number of deaths. There were 147 in this particular week, of which

93 occurred in the white and 54 in the negro population, as contrasted with 79 of the former and 61 of the latter in the previous week. There were 10 deaths in infants. There was a fairly sharp drop in the number of deaths reported for the next week as only 130 deaths were listed, divided 78 white and 52 negro. Only 12 infants succumbed in this period of time. The health of New Orleans was even better than the previous week when the June 3 figures were reported. In this week there were only 120 deaths, 75 in the white and 45 in the negro section of the population. Fourteen infants under one year of age died this week.

INFECTIOUS DISEASES IN LOUISIANA

Dr. J. A. O'Hara, epidemiologist for the State of Louisiana, has furnished us with the weekly morbidity reports for the state, which contain the following summarized information: For the week ending May 20, syphilis, according to its happy custom, led all reported diseases with 127 cases listed. Other diseases occurring in numbers greater than 10 included measles with 67 cases, pneumonia with 44, whooping cough with 42, pulmonary tuberculosis with 29, cancer with 23, hookworm infestation and chickenpox with 20 each, scarlet fever with 16, gonorrhea and typhoid fever with 15 each, and diphtheria with 13. Of the 15 cases of typhoid fever, four were reported from Richland and four from Orleans, one of which was imported. A case of typhus fever was reported from Acadia and one of poliomyelitis from Orleans. Two cases of undulant fever were reported this week. For the twenty-first week, ending May 27, measles sprang to the front as the most frequently reported disease this week; followed by 96 cases of syphilis, 43 each of pneumonia and gonorrhea, 39 of pulmonary tuberculosis, 28 of hookworm infestation, 26 of whooping cough, 18 each of cancer and malaria, 12 of chickenpox and 10 of scarlet fever. Of the unusual cases recorded this week were typhus fever, one case each from Iberia and Orleans, and one case of tularemia, as well as a case of smallpox. For the week which ended in early June, there were recorded 156 cases of syphilis, 79 of measles, 54 of pneumonia, 34 of pulmonary tuberculosis, 27 of malaria, 26 each of hookworm infestation and cancer, 12 of chickenpox and 10 each of typhoid fever and diphtheria. Three cases of typhoid fever were recorded in Bossier this week and two in Concordia. For the week which terminated June 10, syphilis again led all other diseases with 271 instances. The mild epidemic of measles seemed to be subsiding somewhat, only 74 cases being listed this week. The incidence of whooping cough was about three times the five-year average for this disease. There were 63 cases of pulmonary tuberculosis reported to the State Board of Health. This is a larger number than usual, but the number of cases follows more closely to the five-year average than any other. Thirty-nine cases of pneumonia were reported, 35 of malaria, 31 of cancer, 27 of hookworm infestation, 17 of gonorrhea, 11 of diphtheria and 10 of typhoid fever. The typhoid fever cases this week were scattered all over the state. A case of poliomyelitis was found in Acadia and one of tularemia in Caddo. For the twenty-fourth week of the year, ending June 17, there were reported 107 cases of syphilis, 62 of pulmonary tuberculosis, 52 of pneumonia, 29 of cancer, 24 of malaria, 22 of gonorrhea, 14 of measles and 10 of typhoid fever. The only rather unusual disease, but one which is becoming more frequently reported, was undulant fever, with two cases, one each from Avoyelles and Franklin. It is rather interesting that only two cases of pellagra were found in the State of Louisiana, as contrasted with five in the previous week. This apparently is a disease which is diminishing in its frequency. Dengue fever has not been reported for many months. Ophthalmia neonatorum, bubonic plague, rabies, typhus fever and anthrax are only occasionally reported. Bubonic plague is almost unheard of amongst the list of diseases which are required by law to be reported to the State Board of Health.

WOMAN'S AUXILIARY

Louisiana State Medical Society

President—Mrs S. M. Blackshear, New Orleans.

President-elect—Mrs. Roy Carl Young, Covington.

First Vice-President—Mrs. H. O. Barker, Alexandria.

Second Vice-President—Mrs. C. U. Johnson, Monroe.

Third Vice-President—Mrs. W. P. Bordelon, Lake Charles.

Fourth Vice-President—Mrs. T. E. Strain, Shreveport.

Treasurer—Mrs. W. R. Buffington, New Orleans.

Recording Secretary—Mrs. E. C. Melton, Plaquemine.

Corresponding Secretary—Mrs. C. R. Hume, New Orleans.

Parliamentarian—Mrs. A. A. Herold, Shreveport.

Historian—Mrs. Joseph Bath, Natchitoches.

With the recent American Medical Convention still in our minds, we will read with much interest this Convention Letter from Mrs. Frederick G. Ellis, Past President of Louisiana State Auxiliary, also the report of Auxiliary Exhibits from Mrs. Clifford P. Rutledge, President of Caddo Parish Auxiliary.

We are greatly indebted to each of these members for giving us so complete a picture of this important meeting.

NATIONAL AUXILIARY LETTER

Dear Fellow Members:

Once again I am settled into the old rut of household duties after a very hot and strenuous five days in St. Louis at the meeting of the Woman's Auxiliary to the American Medical Association.

The Chase Hotel, headquarters of the woman's division, was a very busy and crowded place as you will understand when I tell you the registration reached an all-time high of 1209 women, 35 of whom were National Board Members, 89 delegates, 22 alternates, 589 members and 474 guests. Louisiana, besides having two members on the National Board, had a full quota of delegates and alternates, a record we have never before attained.

On Sunday, May 14, Mrs. Charles C. Tomlinson, National President, was honored with a tea in the home of Mrs. Willard Bartlett, 53 Westmorland Place, and on Monday morning, May 15, the Board of Directors meeting was held in the Regency Room of Hotel Chase. In the afternoon the guests were taken for a delightful ride and visits were made to some very lovely private gardens, where tea and punch were served.

On Tuesday, May 16, the day started with a Southern breakfast at the Hotel Chase, the President of the Southern Auxiliary, Mrs. Willis K. West of Oklahoma City, presiding and acting as toastmistress. Mrs. West will be remembered by most Louisianians as the former Miss Miriam McGuffin of Shreveport.

From breakfast the members repaired to the Regency Room for the formal opening of the convention. Mrs. Charles C. Tomlinson called the meeting to order and, after the Invocation was given by Rev. Dr. McIvor, introduced Mrs. Willard Bartlett, the general chairman of the convention, who told of the plans made for our pleasure and comfort. The address of welcome and response were beautifully given and were followed by a most fitting memorial service for those members who during the year had joined the hosts in the "great beyond."

After the usual convention roll call the National President read her address in her own pleasing and interesting manner. Reports of officers and committee chairmen filled the balance of the morning. To me the most interesting items were:

1. The growing interest in and the increased subscriptions to Hygeia.

2. The legislative program in conjunction with public relations work in giving lectures and instruction to the members and the lay public on conditions which confront the doctors today should socialized medicine be adopted in this country, and the effect of such legislation on the general public.

3. The membership award went to New York State with 63 per cent increase. Later in the meeting it was voted to discontinue this annual award as the basis of figuring the winner had become too complicated.

4. The Organization Chairman, Mrs. Frank Haggard of San Antonio, only partly finished reading her report, she being allotted ten minutes just the same as all other officers received and decidedly not sufficient time for such an important report. Mrs. Haggard did not get to the Southern states, so please read it when the national books of convention proceedings reach each auxiliary. I am hoping that she left the best till the last for I feel sure the Southern states have done splendidly in this phase of our work.

5. The Chairman of Press and Publicity was not at the convention and I for one was disappointed for her work was especially good in the news letters she so ably edited.

6. Mrs. V. E. Holcombe gave a very comprehensive report of the splendid programs given, based on the monthly material sent from her office. Some phase of health education was used in almost every parish auxiliary program. A number of them, cooperating with the Public Relations Chairman, had open meetings and an outstanding doctor speak on the subject.

At the conclusion of the committee reports the President-elect, Mrs. Rollo K. Packard, was presented to the assembly. Then all adjourned and were taken by bus to the St. Louis Womens' Club where a delightful luncheon was served. This luncheon was in the form of a birthday party at which all past presidents of National were honor guests. Mrs. Hibbitts of Texarkana was a most gracious toastmistress.

Leaving the St. Louis Club again by bus all who wished were taken on sightseeing trips to the many interesting places in the city. Among the points of interest were the art galleries and museum, and all were pleased to have a chance to see the Lindbergh trophies which have been so widely publicised. A stop of one hour was made at the St. Louis Medical Society Building where an elaborate afternoon tea was served by the Woman's Club of the St. Louis University School of Medicine. That night all members and guests were invited to the open meeting of the American Medical Association which was held at the Municipal Auditorium.

Wednesday morning at 9 o'clock the General Session of the Auxiliary was called to order by the president. After the customary routine business was conducted the state presidents were asked to read their reports. Of the thirty-nine states organized only four failed to have a president or member there to read of their work of the past year, and these four reports were filed by the secretary. Many and varied were the activities carried on in each state and a complete report will be printed in the "Minutes and Reports of the National Auxiliary."

Time was called at 1 p. m. for the annual luncheon and the meeting called to order again at the conclusion for the remainder of the business

on hand. The nominating committee presented a slate of officers for the year and all were duly elected as presented by them. The President-elect is Mrs. V. E. Holcombe, Charleston, West Virginia. The incoming President, Mrs. Rollo K. Packard of Chicago, Illinois, was then presented and she and the following officers were installed: First Vice-President, Mrs. David W. Thomas, Lock Haven, Pennsylvania; Second Vice-President, Mrs. Otto C. Hagmeier, Seaside, Oregon; Third Vice-President, Mrs. M. B. Van Cleave, Terre Haute, Indiana; Fourth Vice-President, Mrs. W. K. West, Oklahoma City, Oklahoma; Treasurer, Mrs. E. E. Fisher, Portland, Oregon; Recording Secretary, Mrs. John L. Bauer, Brooklyn, New York; Corresponding Secretary, Mrs. Lucius Cole, River Forest, Illinois.

Thus the open meetings were concluded although Conferences on Public Relations, Program and Hygeia were held. The postconvention board meeting was held on Thursday morning.

At noon on Thursday all who had made reservations for the boat trip on the Mississippi found busses waiting to take them to the dock. We rode in state with motor police ahead, alongside and at intersections, sirens open wide, while six busses made a non-stop trip for us. For three hours we viewed the city from the river and successfully tucked away hot dogs and pop in unlimited amounts. The busses again met the delegates at the dock and drove us back to the Chase Hotel.

Everyone had to hurry and dress for the annual "Bring Your Husband Banquet" and, following this, go to the reception and ball at the Jefferson Hotel, given in honor of the President of the American Medical Society—that is, if the heat and festivities had not been too much for you. I think quite a few of us fell by the way side and I was ready for a wheel chair.

Friday was open to do as we wished and the average person seemed to be anxious to see the elaborate exhibit display. The huge St. Louis Auditorium was filled in every available space with scientific and commercial displays. Only the commercial exhibits were open to the public, the scientific section being open only for doctors and their wives on presentation of the badge.

The Louisianians were housed in widely scattered hotels which was an inconvenience in many ways as it was hard to have the usual "get togethers" always so enjoyable at our state conventions, but when one considers the registration of 7,000 doctors and 1,200 women, one can realize the hugeness of the A. M. A. I, for one, felt like the proverbial small toad in a very big puddle, but I enjoyed the experience of at least being there. The National President, Mrs. Tomlinson, whom we all know personally, was most charming and we all enjoyed meeting her again.

Kindest regards to all of you.

Sincerely yours,

Edna Ellis

(Mrs. Frederick G. Ellis)

4624 Fairfield Avenue

Shreveport, Louisiana

NATIONAL AUXILIARY EXHIBITS

The Exhibits Committee of the Woman's Auxiliary to the American Medical Association, with Mrs. Ily R. Beir of Atlantic City as Chairman, together with the chairmen of the many states sending exhibits, should feel well repaid for the time and thought spent on this contribution to the annual A. M. A. Convention in St. Louis this year. It was the most outstanding array of exhibits yet assembled by the Woman's Auxiliary for several reasons: In the first place, the location was ideal. Adjoining the registration quarters in the beautiful Hotel Chase, the large exhibit room served also as an ante-room to the Auxiliary Convention Hall, where all who attended the meetings must pass through. Also, there were more states represented, as well as more county auxiliaries sending exhibits, which gave a larger and more representative display of activities and progress. States sending exhibits did their work so well that their contributions provided a graphic review of the progress made in auxiliary work, thus furnishing much information and many lessons for officers, delegates and members for the advancement of their own state activities.

The exhibits were well gotten up. Some were beautiful, others novel, and all were of much interest to the eye and brain, and appealed to the imagination. Several hundred visitors registered in the room, and many returned several times to study more carefully the exhibits of especial interest. Brief description of the exhibits by states, are as follows:

Arizona: Map made of beaten copper on a wood base showing the various counties.

Arkansas: Charts illustrating work done for milk fund, prevention of colds, fight on syphilis, better sanitation. Activities: Hygeia essays; research work; health promotion; student loan fund.

California: A banner of velvet and gilt with founding date "1929." Scrapbook of A. M. A. Convention at San Francisco.

Colorado: Panoramic mountain scene showing the different projects on flags carried by mountain climbers.

Florida: Scrapbook.

Illinois: A large, very attractive poster of a sailing vessel "The Good Ship A. M. A." plowing through stormy seas of public opinion toward the port of Better Understanding of the Aims of Scientific Medicine. Chart showing object of Auxiliary: preventive medicine; securing adequate medical legislation.

Indiana: Attractive charts showing work in tubercular hospital, children library.

Kansas: Year book; posters on prevention of colds.

Kentucky: Map and posters, the latter showing home of Jane Todd Crawford, now a memorial library, and Jane Todd Memorial Trail leading to Dr. McDowell's home. Beautifully illustrated book of activities.

Louisiana: Large attractive book made in chart style showing the activities of the organized parishes: Anti-tubercular and anti-cancer drives; Indigent Physicians' Fund; A. M. A. health broadcasts; Doctor's Day; medical samples; Pines Preventorium work among a group of under-privileged children.

Minnesota: Three posters showing power of the doctor's wife in the community, promotion of Hygeia, a county auxiliary's activities; six pictures of other activities. Very active Auxiliary.

Mississippi: Map of State showing counties organized. Scrapbook.

Missouri: Large bound cardboard book with title "The Three Little Pigs and How They Did It," giving the story of how they work and the many activities carried on. Scrapbook; file of clippings.

Michigan: Poster showing six divisions of stressed activities.

New Hampshire: Chart showing organized counties.

New Jersey: Map of State showing organized and unorganized counties. Very complete medical histories. Archives of the Medical Auxiliary.

New Mexico: Indian trinkets. Poster showing work done among Indians.

New York: A unique and outstanding exhibit set in a large box arranged as a stage, with figures in miniature engaged in carrying on the many projects of the different counties.

North Carolina: Map made of wood, showing organized groups by counties.

Oklahoma: Chart showing membership growth of Auxiliary; year book; scrapbook; layette; health posters.

Oregon: Map and models carved out of soap, with cards of explanation.

Pennsylvania: Museum extension project, illustrated by a model. Hygeia. Benevolence Medical Fund which was started in 1927 and now amounts to $31,429.44.

South Dakota: Map of State outlining counties.

Texas: Models, showing a bridge leading from a dilapidated cottage in miserable surroundings to a well kept house set in beautiful grounds. The planks of the bridge which lead to the "better life" bear the names of the Auxiliary projects for advancement in health education.

Utah: A massive book of "Mother Goose on Quacks" rhymes; colorful and clever. Also a very attractive scrapbook.

Virginia: Small dolls, beautifully dressed to represent honored women of different counties. On each was hung a card showing the work done in her county. Layettes. A chart showing names of each county president and exhibit chairman.

Washington: Posters showing a large steeplechase illustrating "Basic Science Hurdles"; "Fight aginst Quacks"; "The Diploma Mill"; a donkey before a radio representing "radio quacker." A splendid exhibit.

Wisconsin: Scrapbooks from many counties; posters on activities; books of State archives; a display by the Visiting Nurses' Association; a massive book containing biographies of physicians from 1838 to 1938; year book. Very large and comprehensive exhibit.

Mrs. Clifford P. Rutledge.

Respectfully submitted,
Mrs. Edgar Burns,
Press and Publicity Chairman.

BOOK REVIEWS

Disorders of the Blood: By Lionel E. H. Whitby, C. V. O., M. C., M. A., M. D., (Cantab.) F. R. C. P. (Lond.), D. P. H., and C. J. C. Britton, M. D., (New Zealand), D. P. H. Philadelphia, P. Blakiston's Son and Co., 1937. Pp. 582. Price $7.00.

In this second edition of Whitby and Britton's valuable work, many advances are reflected, in extra pages, new illustrations and changes in bibliography. The references are largely key papers, many by American writers. The authors stress the value of classifying the anemias in terms of cell size and hemoglobin concentration, as being more reliable and of more practical value than on the basis of color index. They emphasize how rare is primary disease of the hemopoietic system and how often changes in the peripheral blood are a symptom of a disease or disorder in some other system of the body.

The content of this most excellent book covers all phases of the origin, development, function and the fate of the cells of the blood. Chapters are devoted to the causes of anemia, nature and mode of action of the hemopoietic substances, a description of the hemolytic and dyshemopoietic anemias, purpuric and hemorrhagic diseases, the leukemias and disorders of the blood and associated miscellaneous conditions such as splenomegaly and Hodgkin's disease. One chapter is devoted to the anemias of infancy and childhood and more than fifty pages

cover technic, as applied to the study and diagnosis of disorders of the blood.

Within the past few years there has been considerable interest in the subject of anemias coincident with the recent discoveries concerning the etiologic factors involved, notably disorders of the blood due to the newer chemicals.

The book is written in a pleasing style and is replete with valuable plates. As a text for students and practitioners it can be highly recommended as conservative, authoritative and comprehensive.

ELIZABETH BASS, M. D.

Surgery of Oral and Facial Diseases and Malformations: By G. v I. Brown, D. D. S., M. D., C. M., F. A. C. S. Philadelphia, Lea and Febiger, 1938. Pp. 778. Price $10.00.

In this revised fourth edition the author has eliminated many of the earlier procedures which have passed into disuse and has included later basic procedures of proved merit. The book is adaptable as a ready reference both to the general practitioner and the specialist.

One is impressed, however, by the fact that too many conditions have been considered to permit a complete and full description of many of the conditions presented, thus detracting from its value as a text for students. Bibliographies are notably lacking. The value of this edition could have been enhanced by enlarging the section on wounds of the mouth and that on burns and scalds. The chapters dealing with diseases and surgical conditions of the bones of the face and jaws and congenital deformities of the face are particularly good.

The book is adequately illustrated, and is valuable as a ready reference.

NEAL OWENS, M. D.

Concepts and Problems of Psychotherapy: By Leland E. Hinsie, M. D. New York, Columbia University Press, 1937. Pp. 199. Price $2.75.

In a delightful little volume the author presents a discussion of current forms of psychotherapy. Attention is given to psychoanalysis, to the individual psychology of Adler and to the analytic psychology of Jung. In some forty pages the author gives a splendid presentation of the concepts and methods of psychobiology and the methods of therapy utilized by Adolph Meyer and his group.

The advantages and disadvantages, the indications and contraindications of the various forms of psychotherapy are pointed out. A statistical analysis of the various methods of psychiatric treatment is presented. A complete bibliography concludes the work.

This book should find a welcome place in the library of any physician interested in the treatment and care of any of his patients' personality problems. It is unreservedly recommended and it presents in a clear fashion, devoid of any "scientific jargon," many of the facts of psychotherapy of which the physician is constantly in need.

JOHN W. BICK, M. D.

Refraction of the Eye: By Alfred Cowan, M. D. Philadelphia, Lea & Febiger, 1938. Pp. 319. Price $4.75.

It is a pleasure to review this excellent work on an important, but neglected branch of ophthalmology. Its lack of glamour is reflected in the apathetic interest of the average oculist. The relatively rapid rise of optometry and corresponding decline of ophthalmology in refraction tells its own story.

Dr. Cowan has presented physics of lights and lenses in a thorough and comprehensive manner. The laws of refraction and reflection, as well as their applications in every day practice, are written in a very readable way. Technical subjects like the theory of Gaus, the Gulstrand schematic eye, the reduced eye of Donders, the determination of cylinders at oblique axis are presented in an exceptionally lucid style. Like the details of anatomy, they must be learned and forgotten at least seven times before they permanently stick to the memory. Even then, their adhesion in the association centers is usually lightly mucilaginous.

The chapter on visual acuity, like the first line of the, "Star Spangled Banner," is very illuminating.

The chapters on accommodation and ametropia, which together, cover about 50 pages, could possibly have been amplified, as could the technic of practical refraction. They present far more of the subject, however, than 99 in 100 ophthalmologists know. In explaining the cross cylinder, the author has very properly mentioned that it changes the cylindrical component twice as much as the spherical component. The cross cylinder would be used much less frequently if its users knew its resultant combinations.

A good retinoscopinist is usually a good refractionist. The author has detailed its fundamentals and modern applications: cylinder, streak, and dynamic retinoscopy. Among the subjective methods of testing, cyclodamia, velonoskiascopy, and duochrome tests, are elucidated.

Timely is the author's presentation of contact glasses and telescopic spectacles. The publicity which has been given these novelties increases the ophthalmologist's responsibility that his patients do not buy wheelbarrows thinking they are purchasing Cadillacs.

May the second edition of this excellent work appear shortly.

CHARLES A. BAHN, M. D.

Superfluous Hair and Its Removal: By A. F. Niemoeller, A. B., M. A., B. S. New York, Harvest House, 1938. Pp. 155. Price $2.00.

"Superfluous Hair and Its Removal" by A. F. Niemoeller is a 155 page discussion of causes and methods of removal of superfluous hair. The text is written for the lay person, and even goes to the point of recommending those who are not medically trained operators, such as one member of the family, operating an inexpensive apparatus on another member of the family. It does recommend against quack methods of hair removal. The average text on skin diseases, however, contains, in brief, all the medical man need know about causes of superfluous hair and its removal.

M. T. VAN STUDDIFORD, M. D.

A Manual of Fractures and Dislocations: By Barbara Bartlett Stimson, A. B., M. D., Med. Sc. D., F. A. C. S. Philadelphia, Lea & Febiger, 1939. Pp. 214. Price $2.75.

The author of this book had a good motive in writing it. Such a mass of material is found in big texts on fractures that time available for the student to devote to a special type of surgery in his busy course is too little to permit him to grasp the essentials from these texts. This book is "designed as a guide to the wealth of material that is published in textbooks and articles which bewilder the student with their mass of detail." This is a very short text and necessarily its scope is very limited. The discussion of each fracture is divided under the following headings: occurrence, displacements, diagnosis, pathology, treatment, time of immobilization, prognosis and summary. Students may find it a helpful guide or an introduction to the study of fractures, but the material contained herein is too cursory to be relied upon for either school examinations or practice. For example, two pages are devoted to a discussion of treatment of fractures of the hip, that is, reduction and application of a cast, and one additional paragraph is devoted to a statement without a description of internal fixation for fracture of the neck of the femur. Interesting tables in the volume are the percentages of occurrence of fractures, that is, the relative percentage of all fractures which any special injury may cause. The illustrations, all drawings, are simple and instructive. The inevitable Bryant's triangle and Nélaton's line are illustrated. How unhelpful are these two! Obsolete, they still recur in text after text with emphasis enough to make them seem important because the student must know how to answer some questions put to him by either an inexperienced or casual observer who has never discovered that they are usually not helpful,—that they are unreliable and simply vestiges of pre-roentgen days.

HOWARD MAHORNER, M. D.

PUBLICATIONS RECEIVED

The American Journal of Cancer, New York City: Cutaneous Cancer and Precancer by George M. Mackee, M. D., and Anthony C. Cipollaro, M. D.

Harvest House, New York City: Short Stature and Height Increase by C. J. Gerling.

Alfred A. Knopf, Inc., New York City: Sensible Dieting by William Engel, M .D.

Lea & Febiger, Philadelphia: Laboratory Manual of the Massachusetts General Hospital by Francis T. Hunter, M. D. Heart Patients: Their Study and Care by S. Calvin Smith, M. D., Sc. D.

J. B. Lippincott Company, Philadelphia: Diseases of the Nose and Throat by Charles J. Imperatori, M. D., F. A. C. S., and Herman J. Burman, M. D., F. A. C. S. The New International Clinics, Volume II, edited by George Morris Piersol, M. D.

The Macmillan Company, New York City: Medical Microbiology by Kenneth L. Burdon, Ph. B., Sc. M., Ph. D.

The C. V. Mosby Company, St. Louis: The Life and Letters of Dr. William Beaumont by Jesse S. Myer, A. B., M. D. Practice of Allergy by Warren T. Vaughan, M. D.

Stanford University Press, Stanford University, California: Cancer Handbook of the Tumor Clinic, Stanford University School of Medicine, edited by Eric Liljencrantz, M. D.

W. B. Saunders Company, Philadelphia: An Introductory Guide to Biochemistry by Sidney Bliss, Ph. D. Menstrual Disorders by C. Frederic Fluhman, B. A., M. D., C. M. Endocrinology in Modern Practice by William Wolf, M. D., M. S., Ph. D. Health Officers' Manual by J. C. Geiger, M. D., Dr. P. H., Sc. D., LL. D. Medical Jurisprudence and Toxicology by William Dr. McNally, A. B., M. D. A Textbook of Clinical Neurology by Israel S. Wechsler, M. D. A Textbook of Surgery by American Authors, edited by Frederick Christopher, B. S., M. D., F. A. C. S.

Charles C. Thomas, Springfield and Baltimore: Rural Medicine: Proceedings of the Conference held at Cooperstown, New York, October 7 and 8, 1938.